THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

MARCH, 1933

COMMUNICATIONS

THE OPHTHALMIC LESIONS OF BOTULISM: ADDITIONAL NOTES AND RESEARCH

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OMAHA

FOR many years physicians have known that debilitating ocular symptoms, serious prostrating disorders of the gastro-intestinal tract and even death itself occasionally followed the ingestion of poisoned food. Since van Ermengem's[1] discovery of the bacillus botulinus in 1895, this anaerobic organism or its toxin is always found present in cases of food poisoning accompanied by the usual ocular syndrome. Although our present day knowledge of botulism goes but little beyond that of van Ermengem's time, there is a much greater diffusion of the information. Through the attention and efforts of house-wives, farmers, commercial canners, food chemists, bacteriologists and epidemiologists, botulism has become a rare disease. *The Jl. of the Amer. Med. Assoc.*[2] stated that in the United States and Canada from 1899 to 1928, 165 outbreaks were recorded, with a total of 529 cases. While many of these cases lack authentication by bacteriological and toxicological tests, they are, no doubt, off-set by the unreported typical cases and the undiagnosed atypical ones.

The clinical picture of botulinus intoxication has been set forth by van Ermengem as follows: "Secretory disorders of the gastro-intestinal tract; symmetrical motor paralysis, complete or partial, depending very likely on lesions of the medulla, especially of the nuclei of various cerebral nerves and of the anterior horns of the spinal cord. It is characterized:

1. By an arrest of secretion or by a hypersecretion of saliva and bucco-pharyngeal mucus;
2. By more or less complete external and internal ophthalmoplegia (ptosis, mydriasis, paralysis of accommodation, diplopia, internal strabismus);
3. By dysphagia, aphonia, obstinate constipation and retention or urine;
4. By a general weakening of all voluntary muscles;
5. By absence of fever, but with no disturbance either of general sensibilities or of the intelligence;
6. To this syndrome are added frequent respiratory and circulatory troubles, which can lead to a more or less rapid death through bulbar paralysis;
7. Finally, the characteristic manifestations do not appear earlier than 12 or 24 hours after the ingestion of the food and are often preceded by gastro-intestinal troubles that appear gradually and persist for weeks."

The importance of the ocular symptoms was emphasized by Lancaster[3] who said that "usually the eyes are the first organs to be affected, and in mild cases the paralysis may be limited to the eyes." From the paucity of reports by ophthalmologists it is only reasonable to assume that in most cases the patients were attended by their family physicians. In fact, most of the data pertinent to the ocular symptoms have been furnished by others than ophthalmologists.

van Ermengem's tabulation of the ocular lesions is generally accepted, although others have suggested additional symptoms. Ruge[4] reported one case in which he made a diagnosis of papillo-retinitis. Uhthoff[5] stated that internal ophthalmoplegia is the most frequent cause of the amblyopia or amaurosis occurring in botulism, but that involvement of the optic nerve may figure in the visual reduction in some cases. He added, however, that there is no positive ophthalmoscopic or anatomical proof of lesions of the optic nerve. Dickson[6] said that there may be initial scintillations and dimness of vision, but changes in the retina are rarely found. de Saint-Martin[7] reported four cases of involvement of the optic nerve and retina that persisted for months after other symptoms of the intoxication had subsided.

Soon after the discovery of the bacillus botulinus, Marinesco[8] made histological studies of animals that had been fatally poisoned

with botulinus toxin. He concluded that botulism was a disease of the central nervous system wherein the chief lesions were situated in the ganglion cells of the motor nuclei of the cranial nerves. Essentially similar conclusions were announced by Kempner and Pollack[9] in 1897, by Ossipoff[10] in 1900, and by Römer and Stein[11] in 1904. van Ermengem's summary of the work of these experimenters is briefly as follows: It produces a cloudy, fatty degeneration of endothelium; of the secretory cells of certain glands; of striated muscle fibres; of the anterior horn cells of the cord, pons and medulla; and of the motor ganglion cells in the midbrain. There is an increase of neuroglia and a degeneration of the Nissl bodies.

In 1914, Wilbur and Ophüls[12] in reporting the pathology of a human case, declared that "the nuclei of the ganglion cells are perfectly normal." Since they observed marked thrombosis of the cerebral vessels, they were of opinion that the symptoms of botulism depended upon vascular changes. After extensive experimentation on animals, Dickson and Shevky[13] held that the lesions of botulism were not of central distribution but were peripheral. Edmunds and Keiper[14] concluded from their studies that all essential symptoms of botulism can be explained by a more or less curare-like paralysis of the endings of the motor nerves to the voluntary muscles, including the diaphragm, and by a more or less complete paralysis of the parasympathetic nerve endings.

From the lack of unanimity of opinion regarding the cause of the eye symptoms, one of us outlined a series of experiments on animals with a view to deciding whether or not lesions of the visual pathway result from the toxin of clostridium botulinum. Some of the details of these studies have been published[15] elsewhere, therefore they will be merely summarized in this report. So many different problems presented themselves as the work progressed that it was necessary to restrict the experiments in order to develop a few special features. As one of us had a particular interest in the toxicological aspect of the toxin produced by the bacillus botulinus, a great deal of work was devoted to this phase.

In the experiments the material used was as follows: seven dogs, six cats, nine rabbits, three guinea-pigs, five albino rats, three cocks, and between thirty and forty frogs. The rats, rabbits, guinea-pigs and dogs were injected subcutaneously over the abdomen with botulinus toxin; the cocks were injected in the breast; the cats were used for conjunctival instillation and injection into the anterior chamber; the frogs were turned to account in furnishing nerve-muscle preparations for pharmacological experiments. Other animals not included in these figures were two rabbits, two cats and one dog utilized for the purpose of obtaining

nerve-muscle preparations of the extra-ocular muscles and motor nerves. By using the homologue of the external rectus muscle of a cat this preparation was obtained once but it was not well suited to the purpose. The toxin used in these experiments was supplied through the courtesy of Dr. K. F. Meyer, director of the George Williams Hooper Foundation for Medical Research, University of California, and Professor Robert Graham, Department of Animal Pathology, University of Illinois. The three specific strains of botulinus toxin were provided, namely, types A, B and C. The experiments were conducted in the department of Ophthalmic Research and Pathology of the Medical School of the University of Colorado and the Departments of Histology, Pathology and Pharmacology of the Creighton Medical College.

In the case of the dogs, rabbits, white rats and guinea-pigs, the intoxication was carried to the point of death, either by the primary injection of the toxin or by repeated non-lethal doses. The latter procedure was tried in most instances in order to subject the animals to the intoxication over a longer period of time. In all experimental animals that were studied for histological changes in the eye and brain, the autopsies were performed immediately after death to rule out possible post-mortem degeneration. In many cases where death was imminent, the eyes were enucleated under ether. Even small amounts of the anaesthetic caused death and facilitated the immediate removal of the brain.

As rabbits were found to be more satisfactory than other animals for these detailed studies, a brief résumé of the experiments of this group will be given. In none of the rabbits was excessive lacrimation noted. There was no salivation. Dysphagia was present in all instances in advanced stages of the intoxication. This note was entered in the protocols when mucous râles were heard in the throat of the animal. Dryness of the corneae was noted in all registered members of this group except one, which had no eye symptoms, although it lived for six hours after paralysis of the legs and neck had set in. Three of the rabbits were not considered with the group. Two of them had died during the night and were not fit for histological specimens. The other survived two average lethal doses of the toxin without symptoms and was merely kept for observation of later symptoms, of which none was noted. Of the six rabbits counted in this group, only one failed to show pupillary widening of at least some notable degree.

Chinchilla rabbits of approximately five pounds (2·3 kg.) in weight were used and were checked against normal rabbits of the same age and weight for pupillary measurements. It may be added that the rabbits were all kept in the same degree of illumination prior to and during the studies. One rabbit, or 16 per cent., developed nystagmus and a temporary convergence. Two members

of this group had episcleritis at a late stage. One rabbit showed marked sympathetic irritability during the height of the intoxication. This was manifested by a pronounced mydriasis when the animal was agitated. As soon as it became quiet, the pupils contracted to their previously normal size. There was no instance in which miosis could not be induced by strong light, although the reflex was not visible as such in several cases.

For more than two decades it was generally accepted that all of the symptoms which occur in botulism were chiefly dependent upon destructive lesions in the brain tissue. Although this proposition did not explain all of the symptoms that were noted in certain cases, yet it remained unchallenged for a considerable period. Apparently, but little attention was devoted to the subject for a number of years, at least in regard to investigative work.

Among the first to report upon the pharmacological aspect of botulinus toxin was Schübel[16]. He recorded that a poisoning of the nerve does not occur in from four to eight hours; that the nerve absorbs toxin, if at all, only very slowly. At a later date, however, he stated that he had been able to demonstrate a curare-like action of the toxin in frogs and also degenerative changes in the spinal cord of these animals. Dickson and Shevky[13] observed a marked susceptibility to fatigue in the endings of the motor nerves. In the analysis of their experiments with frogs Edmunds and Keiper[14] concluded that the mechanism between the nerve and muscle was affected; in other words, there was a lesion of the nerve endings.

In our work with this phase of the problem it was surmised that at least some hint of the toxicological action of the toxin of clostridium botulinum might be gained through the agency of the classic nerve-muscle preparations. Accordingly the sciatic-gastrocnemius combination was employed in all these experiments. Toxin of different dilutions with normal salt solution was used for immersion for intervals of 15 minutes; after which the nerve (or muscle) was tested by an electrical stimulus. By means of the usual inductorium apparatus and a writing lever to record responses upon a smoked drum, the preparations could be quickly set up, tried for reaction, and again be immersed in the toxin. A control specimen soaked in normal salt solution was utilized in the same manner for each experiment. The inductorium was set at eight centimetres for nerve stimulation and at three centimetres for direct muscle stimulation. Single shocks of the make-and-break current were used.

The first experiments were made with a ten per cent. dilution of the toxin at a temperature of 15° C. As no effect was produced by this technique, we then repeated the studies at a temperature of 25° C. and with a 35 per cent. solution of the toxin. The effect was striking in that all nerve responses failed after 45 minutes,

even when the strongest current was applied. After repeating this experiment several times and obtaining identical results we were assured that the sciatic nerve in these preparations absorbed toxin very readily when the temperature was kept at 25° C. We then continued the experiment by using dilutions of 10, 15 and 25 per cent. of the toxin, controlling the temperature of the solution by means of a water bath. It was of much interest that the period

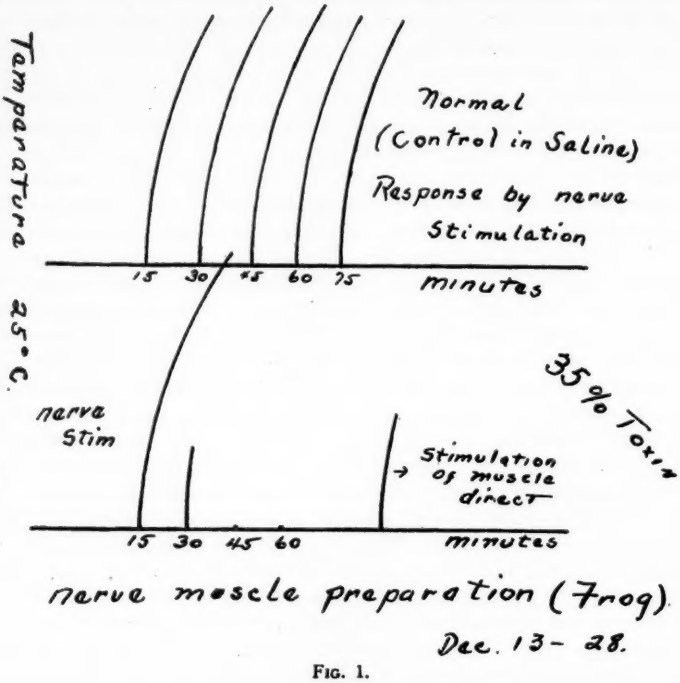

FIG. 1.

Action of toxin on frog nerve-muscle preparation. Upper, saline control; lower, 35 per cent. toxin. Fifteen minute soakings at 25° C. Muscle contraction by electrical stimulation of nerve; single shocks until no response.

of viability of the nerve could be well nigh mathematically calculated, depending upon the strength of the toxin that was used. In the case of the preparation which was soaked in 25 per cent. toxin it was observed that the nerve was not responsive after one hour. Those that had been immersed in 15 per cent. toxin showed no response from nerve stimulation after one and one-fourth hours, while those soaked in 10 per cent. toxin gave no nerve response to the strongest electrical stimulus after 105 minutes.

At the time of lost nerve response it was seen that the muscle would still respond to direct stimulation. By continuing the treatment of the preparation in the toxin, however, a final failure of muscle response was observed. This was almost as striking in its failure to respond as the nerve failure had been, apparently depending upon the dilution of the toxin. Where the preparation was in 25 per cent. toxin the failure of muscle response to the usual stimulus was noted after one and three-quarter hours.

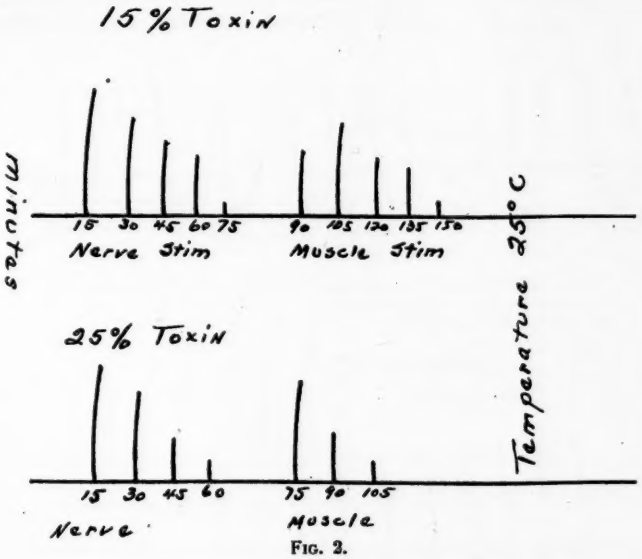

FIG. 2.

Upper figure, 15 per cent. toxin; lower figure, 25 per cent. toxin. Fifteen minute soakings at 25° C. First, contraction of muscle by electrical stimulation of nerve until no response, then electrical stimulation of muscle until no response.

When 15 per cent. toxin was used, the loss of muscle response occurred in two and three-quarter hours. However, in increasing the strength of the electrical stimulus, that is, by setting the inductorium at zero, the muscle gave a weak reaction for some time before total failure could be demonstrated. In most instances the controls were unaffected by the time element and did not exhibit fatigue after the stimulus was increased. There were a few occasions when fatigue was recognized as a factor in the results; namely, when the frogs had been kept so long that they had become inactive.

It was assumed from the frog experiments that the toxin of

clostridium botulinum is a protoplasmic poison as regards peripheral nerve and striated muscle tissues, being apparently more selective in its effect upon the former. Upon arriving at this assumption we endeavoured to anticipate and dispose of the objections to the protoplasmic poison theory of the toxin. The toxin

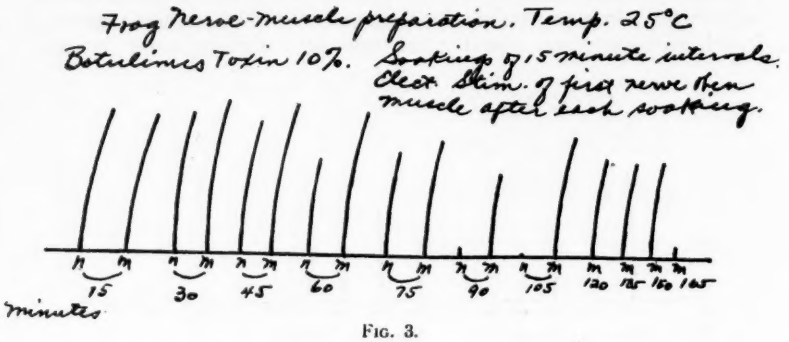
Frog Nerve-muscle preparation. Temp. 25°C
Botulinus Toxin 10%. Soakings of 15 minute intervals.
Elect. Stim. of first nerve then muscle after each soaking.
N M N M N M N M N M N M N M M M M M
15 30 45 60 75 90 105 120 135 150 165
minutes

FIG. 3.

Ten per cent. toxin, 15 minute soakings at 25° C. N.: contraction of muscle by electrical stimulation. M.: contraction of muscle by direct electrical stimulation.

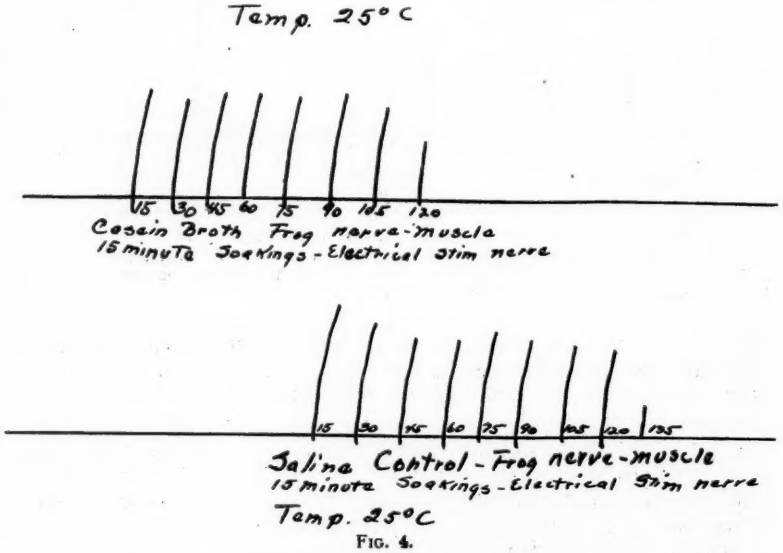
Temp. 25° C
15 30 45 60 75 90 105 120
Casein Broth Frog nerve-muscle
15 minute Soakings - Electrical Stim nerve
15 30 45 60 75 90 105 120 135
Saline Control - Frog nerve-muscle
15 minute Soakings - Electrical Stim nerve
Temp. 25°C

FIG. 4.

Upper figure, casein broth media; lower figure, saline control. Fifteen minute soakings at 25° C. Contraction of muscle by electrical stimulation of nerve.

used in the studies with frogs was type A, which had been furnished for the purpose by Dr. K. F. Meyer. It was prepared in casein digest media and contained no preservative. When used in these experiments the toxin possessed a minimal lethal dose (for a 250 g. guinea-pig) of 0·1c.c. in 36 hours. The hydrogen-ion concentration of the toxin was seven as compared with eight of normal salt solution. Although there was slight fluctuation of the pH, as is to be expected, this factor was reasonably constant throughout the

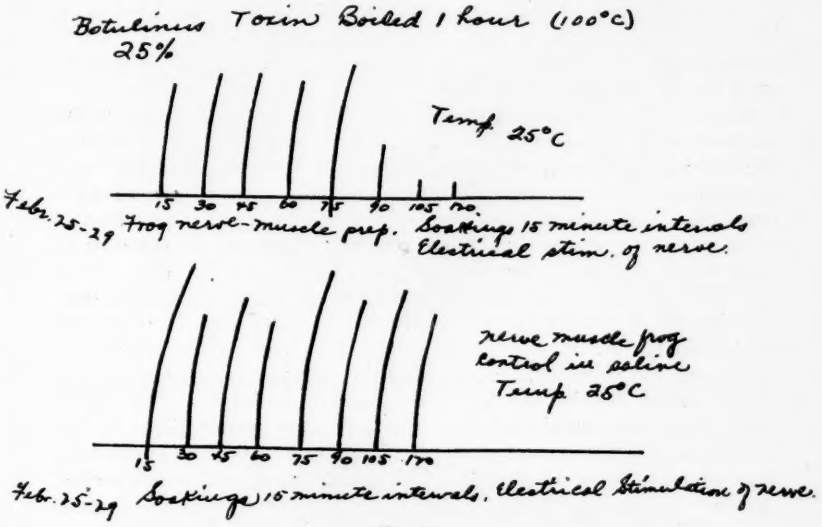
Botulinus Toxin Boiled 1 hour (100°C)
25%
Temp 25°C
15 30 45 60 75 90 105 120
Feb. 25-29 Frog nerve-muscle prep. Soakings 15 minute intervals
Electrical stim. of nerve.
nerve muscle prog
control in saline
Temp 25°C
15 30 45 60 75 90 105 120
Feb. 25-29 Soakings 15 minute intervals, Electrical Stimulation of nerve.

FIG. 5.

Upper, 25 per cent. toxin heated at 100° C. for one hour. Fifteen minute soakings at 5° C. Muscle contraction by electrical stimulation of nerve: lower figure, saline control.

studies, as frequent determinations showed. In order to assure accuracy upon this point, casein digest medium was also obtained from Dr. Meyer. Then by using this broth in normal salt solution corresponding to the dilutions of the toxin that had been employed, it was determined that even in the stronger solutions the nerve-muscle preparations gave adequate response to the usual stimulation after two and one-half hours. This experiment removed the possible objection that the previously recorded results had been caused by the protein in the media. Since the hydrogen-ion concentration of the toxin and that of the media alone were identical, the last experiment also disposed of the objection that the results were dependent upon that factor.

The studies of numerous investigators who have worked upon this phase of the question reveal a marked variation in the heat resistance of the toxin of clostridium botulinum. van Ermengem[17] reported that a temperature of 58° C. for three hours materially weakened the toxin, while a temperature of 80° C. for one-half hour was sufficient to render it almost completely inert. He goes on to state that at 100° C. the toxin was completely destroyed in ten minutes. Shippen[18] working with the Nevin, type B, strain of toxin found that it was destroyed by a temperature of 65° to 70° C. in 20 minutes. Thom, Edmondson and Giltner[19] reported type B. toxin destroyed in ten minutes at 70° to 73° C. Orr[20] investigated ten strains of botulinus toxin which were rendered inert in five minutes at 80° C.

For our experimental studies of the heat resistance of the toxin we endeavoured to make the destruction complete. Accordingly, the toxin was heated to a temperature of 100° C. and this was maintained for one hour. White mice which were injected with one-half c.c. of this mixture showed no effect in four days whereas mice of the same size had been killed in from two to three days with 0·001 c.c. of the toxin before it was treated by heat. This test with living animals was considered as fair evidence of destruction of the toxin by heat. (It seems deserving of comment that Dr. K. F. Meyer[21] had writen that the toxin, which we employed for these studies, showed the greatest resistance to heat of any strain that he had been privileged to examine.) The heated toxin was then tested with frog nerve-muscle preparations by the technique previously detailed. A 25 per cent. solution of the heat treated toxin in normal salt solution was used and was checked against a control in normal salt solution. This study showed that while the toxicity was greatly lowered, it did not seem to be entirely lost. There was a diminution of nerve response in two hours, while the control was normal. Repetitions of this experiment gave almost identical results. If there were any break in our technique which might explain the presence of any toxic substance in these boiled solutions, we have failed to detect it. We offer no explanation of this phenomenon as we ourselves are not entirely satisfied with this result. We are continuing to study this problem with a view to reporting our final opinion at a later date.

The histological changes in the nuclei of the third and fourth cranial nerves were: round cell infiltration; lymphoid cells packed into the parenchyma; extravasation of red blood cells; distention of capillaries with erythrocytes; stagnation of blood; migration of lymphoid cells; thickening of capillary endothelium; neuronophagia, chromatolysis, satellitosis, necrobiosis, nuclear displacement, nuclear shrinking, vacuolation, powdery granulation of Nissl bodies and complete disintegration of the ganglion cells;

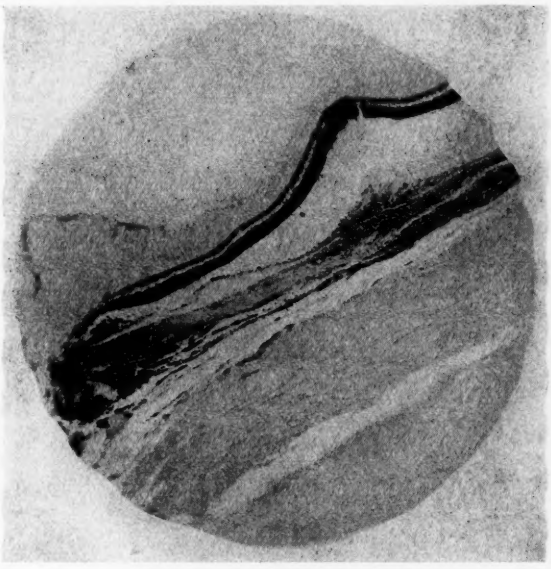

FIG. 1.

Low power, massive interrupted round-cell infiltration in the choroid (× 95).

FIG. 2.

Perivascular infiltration of the iris (× 200).

FIG. 3.

High-power view of degenerating ganglion cells of the retina (× 1,750).

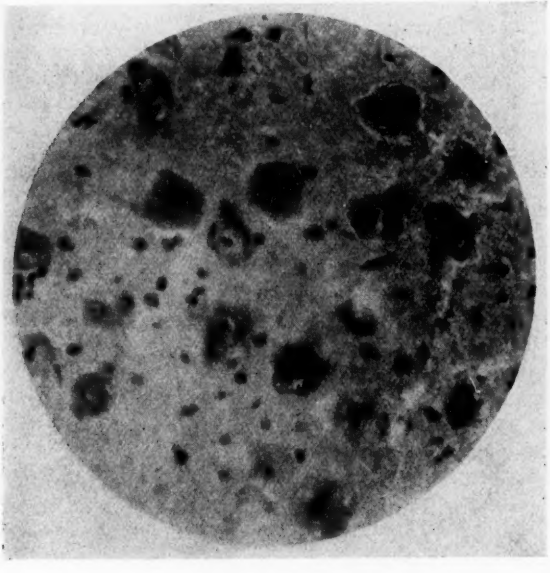

FIG. 4.

High-power view of degenerating nerve cells in the midbrain (× 500).

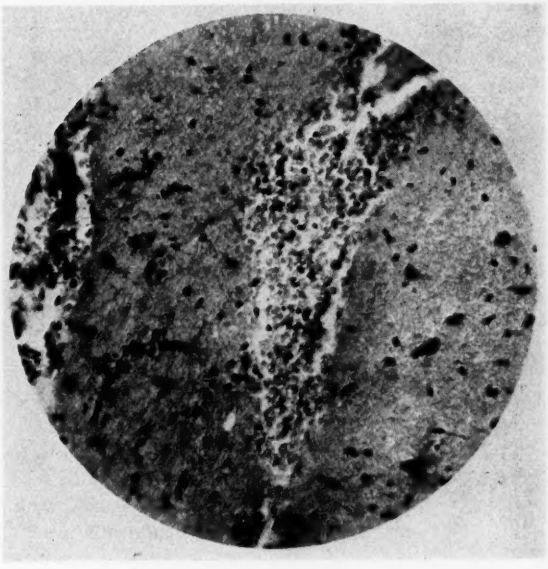

FIG. 5.
Haemorrhagic lesion in the midbrain (× 300).

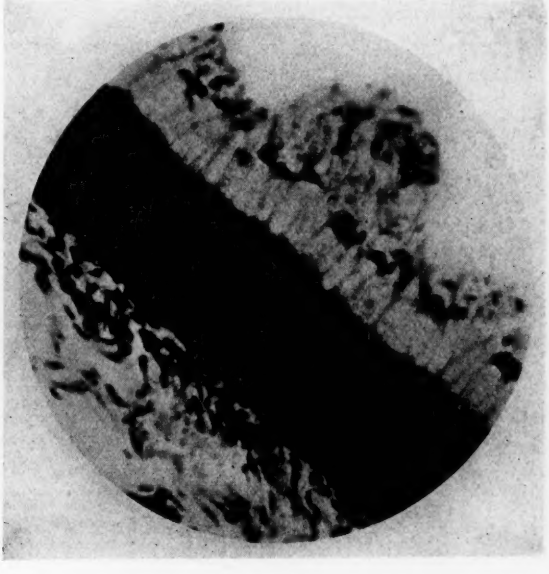

FIG. 6.
Disintegration of the retina (× 400).

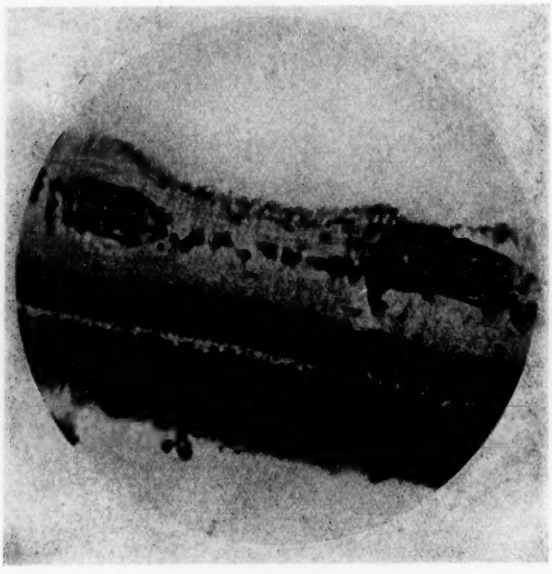

FIG. 7.

Capillary dilatation in the ganglion cell layer of the retina (× 300).

FIG. 8.

Perivascular infiltration in the meninges of the midbrain (× 90).

increase of neuroglia. Similar changes were observed in other parts of the midbrain. Besides a diffuse small round cell infiltration beneath the ependymal lining of the third ventricle, there were diffuse round cell infiltration and massive extravasation of erythrocytes in the meninges. The meningeal vessels were distended with red corpuscles. Thrombosis was not frequent in the midbrain. The changes in the optic nerves were: focal infiltration in the parenchyma, diffuse increase of neuroglia, and round cell infiltration of the pial and arachnoidal sheaths. The changes in the optic tracts were: round cell infiltration, extravasation of erythrocytes, emigration of lymphoid cells and stagnation of blood. The chiasmal changes were maximal infiltration, excessive packing of lymphoid cells in the parenchyma and extensive extravasation of red cells. The retinal changes were: fat formation in the ganglion cell layer; pyknosis, chromatolysis and vacuolation of the ganglion cells; a powder-like reduction of the pigment granules; engorgement of the vessels with red corpuscles and stagnation of blood. In the choroid there was maximal infiltration involving all layers. There was round cell infiltration of the corneo-scleral junction. In the ciliary body there was round cell infiltration together with an increase in the connective tissue element.

The exudative lesions consisted chiefly of lymphocytes and monocytes, some of which had differentiated into polyblasts while others had become transformed into plasma cells. The exudate occurred for the most part about the vessels, but in many instances there was a tendency to migration into the parenchyma. Where maximal infiltration was noted as many as 15 rows of lymphoid cells were present around the vessels.

Findings

Though it is not deemed expedient to draw conclusions that are not fully supported by the present research, as some of the problems mentioned have not received sufficient consideration, the multiplicity of the lesions, together with the severe character of cell destruction and the round-cell infiltration, speaks for the overwhelming virulence of the toxin of clostridium botulinum.

This paper may be presumed to support the work of the European physicians in regard to their histological findings in the nuclei of the cranial motor nerves.

Botulinus toxin is a protoplasmic poison to peripheral nerve and striated muscle tissues, although more selective in its action upon the former.

Since the toxin of clostridium botulinum is a protoplasmic poison to peripheral nerve and striated muscle and since it produces marked degenerative changes in the central nervous system and

also in all the tunics of the eye, it is possible and even probable that it is a general protoplasmic poison.

The theory of botulinus toxin acting as a general protoplasmic poison would explain all of the lesions herein and heretofore recorded as findings in established botulinus intoxication.

That botulinus toxin may contain a substance of exceptional heat resistance, when judged in the main by delicate frog nerve-muscle experiments, is apparent from this study. More detailed work will have to be done in this field however, before the above statement can be advanced with any degree of assurance or finality.

REFERENCES

1. **van Ermengem, E.**—*Arch. de Pharmacol.*, Vol. III, p. 213, 1897.
2. Special Article.—The Frequency of Botulism. *Jl. Amer. Med. Assoc.*, Vol. XC p. 764 (March 10), 1928.
3. **Lancaster, W. B.**—*Trans. Amer. Ophthal. Soc.*, Vol. XIV, p. 648, 1916; *Ophthalmoscope*, Vol. XIV, p. 588, 1916.
4. **Ruge, S.**—*Klin. Monatsbl. f. Augenheilk.*, Vol. XL, p. 408, 1902.
5. **Uhthoff, W.**—*Graefe-Saemisch-Hess Handbuch*, edition 2, Vol. XI, p. 164.
6. **Dickson, E. C.**—Monograph No. 8, Rockefeller Inst. Med. Res., 1918.
7. **de Saint-Martin. R.**—*Bull. et mém. Soc. Méd. d. hôp. de Paris*, Vol. XLIV p. 52, 1920.
8. **Marinesco, G.**—*Presse Méd.*, Vol. V, p. 41, 1897.
9. **Kempner, W.,** and **Pollack, B.**—*Deutsche med. Wochenschr.*, Vol. XXIII, p. 505, 1897.
10. **Ossipoff, V. P.**—*Ann. de l'Inst. Pasteur*, Vol. XIV, p. 769, 1900.
11. **Römer, P.,** and **Stein, L.**—*Arch. f. Ophthal.*, Vol. LVIII, p. 291, 1904.
12. **Wilbur, R. L.,** and **Ophüls, W.**—Botulism. *Arch. Int. Med.*, Vol. XIV, p. 589 (October), 1914.
13. **Dickson, E. C.,** and **Shevky, R.**—*Jl. Exper. Med.*, Vol. XXXVII, p. 711, 1923.
14. **Edmunds, C. W.,** and **Keiper, G. F.**—Action of Botulinus Toxin. *Jl. Amer. Med. Assoc.*, Vol. LXXXIII, p. 495 (December 20), 1924.
15. **Swab, C. M.**—*Trans. Amer. Ophthal. Soc.*, Vol. XXVII, p. 383, 1929; *Amer. Jl. of Ophthal.*, Vol. XII, p. 949, 1929; *Arch. of Ophthal.*, Vol. III, p. 437, 1930.
16. **Schübel, K.**—*Arch. für Exper. Path. und Pharmak.*, Vol. XCVI, p. 193, 1923.
17. **van Ermengem, E.**—*Zeitschr. f. Hyg.*, Vol. XXVI, pp. 1–56, 1897.
18. **Shippen, L. P.**—*Arch. Int. Med.*, Vol. XXIII, p. 346, 1919.
19. **Thom, C., Edmondson, R. B.,** and **Giltner, L. T.**—*Jl. Amer. Med. Assoc.*, Vol. LXXIII, p. 907, 1919.
20. **Orr, P. F.**—*Jl. Med. Res.*, Vol. XXXVII, p. 127, 1920–21.
21. **Meyer, K. F.**—Comment in Personal Communication.

LOCALIZATION ON THE FUNDUS; CONTRIBUTIONS ON MERIDIAN FAULTS; A NEW LOCALIZING PERIMETER

BY

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IN the treatment of detachment of the retina, the operation of Gonin (hole closing method) has opened a new era. Subsequently two other variations of his method (chemical-caustic and diathermic) have been developed. For each of these three methods the localization of the hole in the retina is of the utmost importance.

In most cases of detachment we find one or more holes in the retina. According to Gonin these holes play a decided rôle in the origin of the detachment. According to this supposition, if we can succeed in occluding these holes, the disease will be healed and the retina reattached. The occlusion of the hole is caused by means of a thermocautery, after the position of the hole has been determined. As we see it, an important feature of the operation —apart from the technique—is the localization. This consists of two parts: *viz.* the localization of the pathological changes of the fundus, the tear in the retina; and further, the marking of this retinal tear on the outer surface of the sclera at the point where the operation must be performed.

The determination of the spot on the globe demands two data, the meridian and the latitude. We determine the meridian by estimation according to the numbering of a watch and mark it on the limbus at the point opposite the tear. The determination of the latitude is made as follows: we take the most peripheral part of the fundus visible by ophthalmoscopy on the fully dilated pupil for the ora serrata and from this point we measure the distance between retinal tear and ora serrata in disc diameters. We regard the distance of the ora serrata from the limbus as 8 mm. and to this we add the measurement of the disc diameters, 1 disc diameter = 1·5 mm., which we transfer by means of dividers to the sclera.

The method based upon estimation is simple for the experienced, but where the necessary experience is wanting, the estimation very often gives incorrect results. In cases in which the estimation was right, but the result was not controlled by means of other methods, the surgeon does not have the feeling of certainty which the use of these other methods would have supplied.

In some essential points the localization on the perimeter is similar to the above mentioned method. In this case we also determine the meridian and the latitude, with the difference, that in

this method the distance is not *estimated* from the ora serrata, but *measured* on the perimeter. In this case, the meridian is determined by the angle which the plane of the perimeter arc forms with the horizontal, and the latitude by the angle which is formed by the line in which the ophthalmoscopic examination takes place and the line of sight of the eye to be examined. The size of the angle found on the perimeter we transfer to the eye to be operated upon in the following manner. In the determination of the latitude we must know what distance on the eye corresponds to the value of the angle read on the perimeter. Concerning this we find numerous data in the literature. Table I shows the chord distances in millimetres, measured from the limbus, which correspond to the angles on the perimeter arc.

TABLE I (according to Weve).

The chord-distances from the limbus (in mms.) referring to the angle values of perimeter arc.

Angle	Donders	Druault	Hallide	Colenbrander	Weve	Phantom eye
90°	8·0		7·8	7·25		7·0
80°	9·4	9·7	9·5	9·15	8·5	8·75
70°	11·4		11·4	11·5	10·5	11·0
60°	13·35	13·4	13·4	13·2	12·75	13·0
50°	15·5		15·2	15·4	14·5	15·0
40°	17·0	17·2	16·8	17·4		17·0
30°			18·3	19·15		19·0
20°		20·4	19·5	20·75		21·0
10°			20·6	23·1		

Some of the authors found their data in an empiric manner; they examined exophthalmic patients or newly removed eyeballs; while others determined geometrically the length of the chords according to the corresponding latitude angle measured on the perimeter arc. As we see by the table, the measurements and reckonings give almost an identical result. By means of the table we can, therefore, find in a simple manner the value of the angles of latitude measured in millimetres from the limbus.

The transfer of the meridian to the eye to be operated upon is theoretically easy. We mark two opposite points on the limbus on the horizontal diameter, and we measure or estimate the operating meridian with regard to the horizontal one. Before reviewing the methods based upon the perimeter we shall refer shortly to the difficulties of the determination of the meridian and the rôle which meridian mistakes play.

In the literature many authors desire the most exact determination possible of the meridian, but there are some who pay less attention to the matter.

According to Clausen ". . für den Erfolg der Ignipunktur ist die genaue Deckung des Lokalisationsmeridians mit dem Netzhautriss wichtiger als ein kleiner Irrtum in der Bogenlänge." In opposition to that Colenbrander says: "Das Bestimmen des Meridians macht keine Schwierigkeiten. Dazu sind keine Berechnungen nötig. Eine gewissenhafte *Abschätzung** gibt hier die besten Resultate . . ." Some authors particularly emphasize the fact that the fault, caused by the mistaken determination of the meridian is greater the more distant the place to be operated upon is from the limbus. *E.g.,* Weve writes ". . bei weit vom Limbus entfernten Rissen ein kleiner Fehler in der Meridianbestimmung üble Folgen haben kann." We might conclude that faults in connection with determinations nearer the limbus play a less important rôle.

We have not found any statistical data covering this matter. The general view is that smaller faults in localization do not count, because the area of cauterization is so large that the tear falls within its radius. As this question has not been settled we made reckonings in order to determine how exactly we must mark the spot of cauterization, *i.e.,* how great exactness a successful Gonin operation requires. The essential part of the question is: what errors might be caused by faults in determination of the meridian, or by its transfer to the eyeball, and whether the errors caused by faulty meridians are greater than the area of cauterization. The question is, therefore, in case the determination of latitude is exact, what degree of meridian fault might cause an unsuccessful operation?

Those determinations which are based upon estimation, determine the meridian in such a manner that the position of the tear is referred to the edge of the pupil or to the limbus, which are viewed as the face of a watch. The face of a watch being divided into 12 parts, each hour represents 30°. As the size of an angle is the more difficult to estimate the shorter are the angle sides, naturally in such a small circle as that of the pupil or of the limbus we can easily make mistakes. The determination of the meridian on the perimeter has the advantage that it takes place in smaller units and so decreases the possibility of error.

The transfer of the meridian to the eye to be operated upon is very important. Here any mistake concerning the meridian becomes greater the nearer it is to the equator. Before and behind the equator the meridian faults are less, although the differences of

* The *italics* are mine.

values where the operation mostly takes place are scarcely to be taken into consideration. The most important place, from the operation view point, is that between the ora serrata and equator, this being the place where most retinal tears occur. Behind the equator only the first 6 mm. are to be considered, as so few operations are performed on the posterior pole, that no regard will be paid to this point in the following remarks.

We have determined the deviations caused by faulty meridians at 10° and 15°. Ten degrees are equal to one-third of an hour on the face of a watch, and 15° to one-half. If we err in determining the meridian by 10°, the difference from the ora serrata to the equator is equal to 1·88 – 2·09 mm., and at 15° 2·82 – 3·13 mm. In other words we make a deviation of about 2 mm. by an error of 10° or 3 mm. by 15°.

The reckonings were calculated on an eyeball, the corneal diameter of which was assumed to be 12 mm., the radius of the sclera was assumed to be 12 mm. The difference between the radius of the anterior and posterior parts of the globe is negligible in determining meridian faults. The problem, therefore, is that of constructing or reckoning that part, 10° or 15°, of latitude which corresponds to the chord's length. The corneal diameter d, and scleral radius r, and the chord b, are known. The angle a can be determined (Fig. 1).

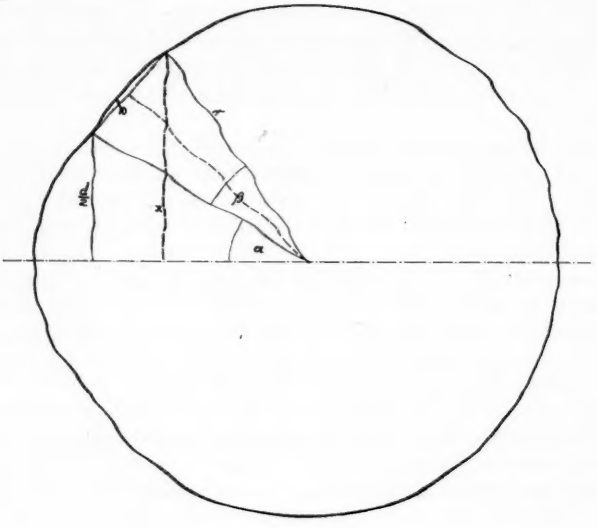


FIG. 1.

$$\sin a = \frac{d/2}{r} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad 1$$

The angle β corresponding with the tendon b is equal to

$$\sin \beta/2 = \frac{b/2}{r} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad 2$$

The radius of latitude circle corresponding to the chord is equal

$$x = r. \sin (a + \beta) \quad \ldots \quad \ldots \quad \ldots \quad 3$$

The arc of 10^0 or 15^0 of the circumference of the circle,

$$\text{radius } x = \frac{2r\pi}{36}; \frac{2r\pi}{24} \quad \ldots \quad \ldots \quad 4$$

shows the corresponding meridian faults.

Fig. 2 gives a graphical explanation.

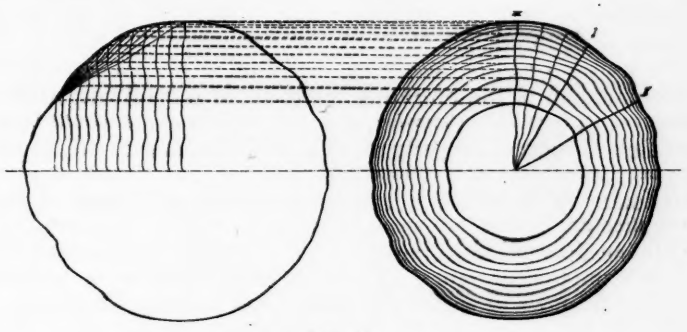


FIG. 2.

In Table II the values are given as far as the equator. The same values hold good for the 6 mm. distance behind the equator, but in an inverted series, *i.e.*, the greater the distance therefrom, the fewer the meridian faults.

TABLE II.

Chord-distance	X	10° meridian fault	15° meridian fault
7 mm.	10·78 mm.	1·88 mm.	2·82 mm.
8 „	11·20 „	1·95 „	2·92 „
9 „	11·54 „	2·01 „	3·02 „
10 „	11·79 „	2·06 „	3·08 „
11 „	11·945 „	2·08 „	3·12 „
12 „	12·0 „	2·09 „	3·13 „

Gonin's operation by cauterization causes the edges of the retinal tear to stick to the choroid by a process of seclusion by means of an adhesive inflammation. (Fisher.) Therefore the greatest success for the operation is assured if the cauterization exactly finds the retinal tear. It is advisable to cauterize both edges of the larger holes. In Luntz's experimental investigations it is to be seen that the area of cauterization is about 3·5 mm. in diameter, *i.e.*, a maximum of 2 mm. in radius. This means that in cases of 10° meridian fault it is probable that the tear does not fall into the area of cauterization, and in cases of 15° meridian fault—where its value is 3 mm.—the area of the cauterization does not even touch the retinal tear.

In reckoning the effect of meridian faults it is to be seen, that the exact determination of the meridian plays an important rôle in the success of the Gonin operation and relatively a small fault in meridian, one-half or one-third hour, makes the success of the operation doubtful.

In transferring the localizing meridian to the globe, *i.e.*, the determination of the operating meridian, it is most important to *measure* it, because otherwise, *e.g.*, by *estimation*, it would be impossible to transfer properly the *exactly determined* meridian to the globe. The method proposed by Lindner for transferring the meridian by means of a goniometer is exact, though a little complicated. Later on we propose a method of projecting the meridian directly on to the eyeball on which we can immediately mark it and thus prevent the sources of error caused by the transfer. From the above remarks, the importance of exact localization is evident. Let us see how far the methods based upon the perimeter satisfy these demands.

The regular perimeter is difficult to use in localization. (1.) Because the supporting arm and its base hinder the ophthalmoscopic examination between 4.30 and 7.30. (2.) We can only perform ophthalmoscopic examination at the edge of the perimeter arc and thus the meridian found is inaccurate, because the scale on the perimeter refers to the centre of the arc. As the retinal tear often occurs in the upper part of the fundus an ophthalmoscopic examination from below is necessary. The following methods are intended to prevent such mistakes.

Lindner's apparatus consists of a perimeter arc mounted on a Gullstrand ophthalmoscope. The examination takes place as follows: The patient looks at the lamp fastened to the perimeter arc and the movable lamp is put in such a position as to bring the retinal tear into the visual field of the ophthalmoscope. Here also the two above-mentioned angles, the meridian and the latitude, have to be determined. According to Lindner, the transfer of the meridian to the eyeball is performed by sewing a small metal

circle—which is divided into 360°—to the globe and the meridian is shown by a hand fixed to the centre of that circle. The use of Lindner's apparatus is limited because some parts of Gullstrand's ophthalmoscope come into collision with it and in these parts the apparatus cannot be used. Another disadvantage of the method is that during the determination, the eye moves and principally in localization, on the extreme periphery the rotation of the eye makes accurate determination of the angle values impossible.

Guist's method may be classed in the same group. Here we determine the two angles above mentioned. The patient constantly looks at one point in the centre of the perimeter arc. The spot on the fundus to be localized we search for by means of a movable ophthalmoscope mounted on the perimeter arc. In comparison with Lindner's method that of Guist has the advantage of determining every spot on the fundus, the perimeter arc being capable of use in ophthalmoscopic examination in every position. As the ophthalmoscopic examination takes place in the axis of the perimeter arc, the finding of the meridian also is exact. Guist's method is reliable but the determination is difficult owing to the cumbersome apparatus.

The apparatus recommended by Weve is a perimeter arc moving on a vertical axis. The determination takes place on a recumbent patient in such a manner that while the patient looks at a small lamp in the centre of the perimeter arc, we first seek the tear indirectly by means of an electric ophthalmoscope on the perimeter arc, afterwards taking away the condenser and looking through the pupil into the eye. The tear is shown by a bright red reflex. The position of the ophthalmoscope then corresponds to the projection of the tear. Now we place a glass eye on the same spot as the eye of the patient previously was. The glass eye we illuminate by aid of the ophthalmoscope from the same spot on the perimeter arc and then find the image of the ophthalmoscopic lamp on the frosted wall of the glass eye, which corresponds to the tear in the patient's fundus which without any further reckoning we may immediately measure.

Some details of determination, as well as the transfer of results on the eye to be operated upon, may easily become sources of error. The substitution, *i.e.*, the placing of the glass eye in the position occupied by the patient's eye, demands special attention. Further delicate points are the measurement of the spot to be localized, which is performed with the aid of a pair of dividers specially designed for the purpose, and the transfer of the result to the patient's eye. There are therefore three such aspects in which mistakes must not occur, *i.e.*, there is a possibility of three sources of error.

F. P. Fischer's localizing ophthalmoscope is not essentially

different from those already mentioned. A Gullstrand hand ophthalmoscope is mounted on the perimeter arc. Great attention must be paid to the adjustment of the apparatus and to the placing of the eye in the curvature centre of the perimeter arc. The determination takes place by means of indirect ophthalmoscopic examination and it refers neither to the nodal points of the globe, (Lindner, Guist,) nor to the plane of the pupil, (Weve,) but to the vertex of the cornea. The results gained are drawn in natural size on a paper and that is placed on the globe to be operated upon, where the point sought for may be immediately found.

Fischer's method cannot be used in some cases and particularly in cases of most peripheral tears because it can be employed only up to 65°. The transfer of the results to the patient's eye takes place essentially in the same manner as Lindner's with the difference that Fischer places the *flat* paper on the *curved* surface of the globe, a method which may give rise to errors. The advantage of the method is exact determination. Fischer's apparatus, from a technical point of view, has remained behind the apparatus already known. (Guist, Weve.) The merit of Fischer's contribution is that he has shown the importance of the fact that the localization must refer to the vertex of the cornea.

Below we shall draw attention to a localizing perimeter which has been employed in our clinic for about three years. In comparison with already described perimeters, although it shows some points of similarity, the simplicity of its treatment, the certainty of its results and finally because of the possibility of direct transfer of the results obtained to the eye, we find it worth while to publish. The model of the apparatus was demonstrated before the *Hungarian Ophthalmological Society* at its annual meeting, 1931.

As we have already mentioned, we have need of such apparatus as will be useful at every point on the perimeter arc, *i.e.*, by which every point of the fundus may be determined. Further, it is necessary that the ophthalmoscopic examinations, take place in the central line of the perimeter arc, *i.e.*, that the meridian may be exactly found. The first condition is fulfilled by the fact that with aid of an articulation, the perimeter arc may be placed in any position so that the supporting arm does not hinder the ophthalmoscopic examination. The second condition is fulfilled by performing the examination in the central line of the perimeter arc.

The perimeter* is mounted on a heavy three-legged stand. (Figs. 3 and 4.) The rectangular arm supporting the perimeter arc can be raised or lowered by means of the cog-wheel. Mounted on the rectangular arm is a joint which enables the perimeter to be turned horizontally in the axis of the arm. This articulation ends in a

* Made by Theodore Hamblin, Ltd., 15, Wigmore Street, Cavendish Square, London, W.1

socket. The axis of the socket and that of the articulation form an angle of 90°. The socket resembles a drum, on the top of which an arc about 25 cm. in diameter is fixed. The perimeter arc is mounted in a rectangular position on the before-mentioned arc. The level of the upper edge of the perimeter arc cuts through the centre of the socket. By means of the joint and the drum-like socket the perimeter arc may be turned in any direction, *viz.*, by aid of its articulation the perimeter arc may be turned downwards (Fig. 4), so that we can examine patients in a recumbent position. The small arc placed within, makes it possible to examine at every point of the perimeter, as the size thereof makes it easy for the surgeon to put his head into it.

A small movable ring-like slide is placed on the 180° perimeter arc, exactly in its middle line and through this the ophthalmoscopic examination takes place. This slide exactly marks the place of the examination. In addition, there is a movable disc on the perimeter arc, which contains the indicators necessary to visual

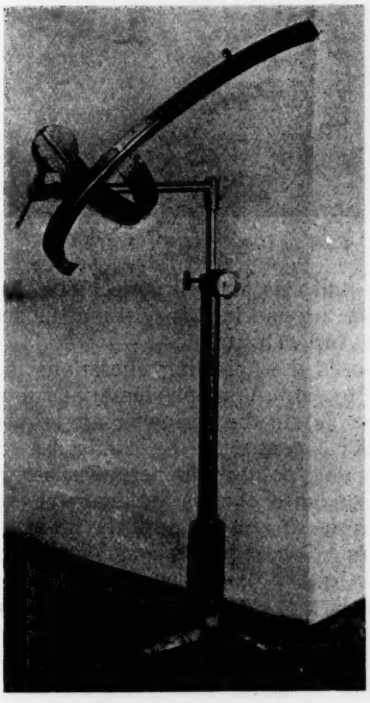


FIG. 3.

FIG. 4.

field examination and also a small lamp. The disc can be moved to the centre of the arc and in this position the small lamp serves as a fixation point in localization.

We have proposed the following alterations in the model now in preparation (Fig. 5). A supplementary arm containing a removable condenser (*c*) on the perimeter arc is mounted in such a way that the optical centre of the condenser falls on the line connecting the turning point of the perimeter arc with its curvature centre (*o*). The condenser throws the image of the small lamp (*l*), which is placed in the turning point of the perimeter arc, on to the curvature centre, *i.e.*, on about 33 cm. This arrangement, which consists of condenser and fixing lamp, plays a double rôle: (1) by means of it we place the eye to be examined in the curvature centre; (2) we transfer the meridian found on to the globe.

The eye must be at the curvature centre of the perimeter arc, otherwise the determination of the angle values will be impossible. The placing of the eye in the curvature centre is assisted partly by means of the condenser, *i.e.*, the image of the fixing

lamp thrown by the condenser falls into the curvature centre. Therefore we must place the patient's eye so that the image of the fixing lamp falls exactly on the cornea. Because of reasons to be mentioned below, the filament of the fixing lamp is a single straight line, the image of which is distinct and the focusing is not difficult. In order to determine when the eye is in the desired position we make use of the finder (*f*) placed on the end of the perimeter arc, which is adjusted on the curvature centre. In this manner we place the eye in the right position by means of a line drawn from the turning point to the curvature centre (*l*-o) and one drawn from the finder to the other end of the perimeter arc (*f*-o-*e*), *i.e.*, its diameter (*f*-*e*).

The above-mentioned condenser also plays a rôle in determination of the meridian. The straight filament of the fixing

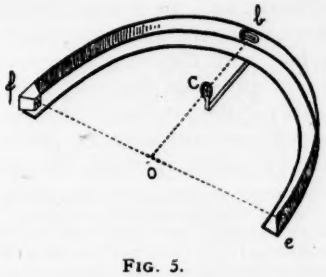


FIG. 5.

lamp is in the level of the perimeter arc and thus follows its revolution, *i.e.*, always marks the position of the perimeter arc in the localization of the meridian. The condenser projects the image of the filament on to the eye and the bright line of light touches the limbus at two opposite points. Therefore we have a means of projecting the meridian on to the eye at the moment of determination.

A plate covered with Stent's wax into which the patient bites, (Beissbrett), which can be raised or lowered at will, or a chin-rest, both of which are fixed to a turn stool, serve to keep the patient's head in the proper position. This attachment makes it possible to examine the patient in the most comfortable position. If the searched for place be in the upper part of the fundus, the raising of the perimeter arc and the biting arrangement makes the examination easier, just as it does in the lowering of the perimeter arc and the biting arrangement in the examination of the lower parts. By this means we can almost entirely avoid inconveniences in the examination.

The separation of the head fixing apparatus and of the perimeter arc we also find in Weve's and Fischer's method. But Weve can examine patients only in a recumbent position, and Fischer's head fixer is mounted on a table. Formerly we also

used the same method, *i.e.*, by placing the patient's head in the chin-rest of the slit-lamp. In our experience, the table between the patient and perimeter proved a hindrance. By mounting the head fixer on a turn stool, the apparatus became much easier to handle. Here we should like to remark that the stool fitted with the head fixer may also be employed in cases requiring Bjerrum's examination, ultra-violet irradiation, also red-free ophthalmoscopic examination, in a word, in all cases where the fixing of the eye is important.

The determination takes place as follows. In a previous examination of the eye we gain a certain knowledge of the fundus and then continue the examination with the aid of the perimeter. We place the patient's eye in the curvature centre of the perimeter arc. For this purpose we roughly measure about 33 cm. distance, the radius of the perimeter arc, with a ruler or thin lath. The more exact adjustment of the eye takes place according to the above mentioned method. Then the patient looks fixedly at the lamp at the turning point of the perimeter arc. The surgeon searches for the tear most expediently with an electric (self luminous) ophthalmoscope in an indirect manner. Then we take away the condenser and search for the tear with the ophthalmoscope. In most cases the tear is easily recognisable because the surrounding grey detached retina is very different from the red reflex of the tear. If the ophthalmoscopic examination is performed through the ring-like slide of the perimeter it makes the findings more exact.

In the first examinations we tried to make the determination by placing a movable arm on the perimeter arc which was parallel with its radius. On this arm a condenser and a crossed thread were mounted. Both were movable on the arm so that the common axis of the cross thread and condenser ran into the curvature centre. We were obliged to give up this method because the exactly regular formation of perimeter arc did not succeed and thus the common axis did not always point towards the curvature centre. Therefore we turned to the method in which a condenser was not used. In the examination we place the plane of the iris, *i.e.*, pupil in the curvature centre and having in view the short distance between the vertex of the cornea and the iris, we may say that the determination took place on the vertex corneae. (Weve.)

The angle found on the perimeter arc gives the latitude, and the position of the arc the meridian. As these two data locate any point of the fundus, the records of results of examinations made at different times become comparable.

From the point of view of operations, the transfer of the results to the globe is important. For this purpose we project the meridian in the before-mentioned manner on to the eye, and mark the two opposite points on the limbus with Indian ink or fluorescein. *Therefore the determination of the meridian, which according to the literature is difficult, is simplified as far as possible by the above-mentioned method.* The latitude reckoned in millimetres from the limbus we can read on the perimeter arc, as the chord distances corresponding to the angles are marked there.

In the former examination the patient looked fixedly at the lamp placed in the centre of the perimeter arc and the ophthalmoscopic examination took place on the arc. If now we draw the ring-like slide to the centre of the arc and the patient looks at the sliding

lamp while we examine the eye through the ring, we can control the results obtained. Here the determination is made as in Lindner's method. In this case the patient's line of sight and the line of ophthalmoscopic examination are changed, *i.e.*, the line of ophthalmoscopic examination comes into the centre of the arc and that of the patient's sight into the same latitude as that in which formerly the examination took place, but at a meridian-angle of 180° further. By this means the determination is not so exact, especially in the extreme positions of the eye, as when the eye is in a position of rest and looking straight in front, but the accuracy is quite sufficient if we use it to supplement data gained by means described above.

With this we have finished the localizing methods based upon the perimeter. Its employment in localization is important because the determination of the meridian and latitude takes place in smaller units and thus the possibility of error is lessened, we project the 12 mm. radius globe on to a 33 cm. radius perimeter and thus we make the determination as if under a magnifying glass.

Imre's method is to be noted separately as it stands between the methods of *estimation* and of *measure*. The meridian is estimated, and the latitude measured by a simple method. We sew a long silk thread into the limbus, at right angles to the meridian of the tear. One end of the silk is put through the hole of the ophthalmoscope and the other end is held by an assistant and serves as a fixation point for the patient's eye. The retinal tear we bring into the middle of the ophthalmoscopic field, and then measure the angle formed by the silk. The advantage of this method is that the determination can be made very quickly and that nothing is necessary except a goniometer.

The common feature of the methods hitherto described is that we obtained either by estimation or by measurement two data, *viz.*, the meridian and the distance from the limbus. By these two data any point on the fundus may be characterized. Majewski and Comberg have tried to localize the position of the tear by transillumination of the sclera. According to them if the light be placed behind the tear, and at the same time we perform a direct ophthalmoscopic examination, the tear in the retina will be shown by a bright spot. By this means the place of the tear answers to the position of the light on the sclera, and thus the place on which the operation is to be performed is immediately found. This method, which seems the simplest, is of no practical value because due to the opacity of the sclera the rays, which pass through it, are dispersed.

Summary

1. We have determined the amount of the deviation caused by faulty meridians with regard to the spot on which most Gonin operations are performed. These faults in cases of error of 10°

are about 2 mm. and in those of 15° are 3 mm. If determination of meridian takes place according to the face of the clock, 10° is equal to one-third, 15° to one-half of an hour. In determinations based on estimation such mistakes may easily occur, and in themselves may make the success of the operation doubtful.

2. The localization on the fundus can be most exactly made with the aid of a perimeter because we determine the meridian and latitude in smaller units and thus lessen the possibility of faults occurring.

3. Besides being easy to manipulate, the proposed localizing perimeter makes it possible to project the meridian on to the eye and thus we can avoid faults connected with the transfer.

LITERATURE.

Clausen.—Ber. d. deutsch. Ophthal. Gesellsch., Vol. XLVIII, p. 63, 1931.
Colenbrander.—*Arch. f. Ophthal.*, Vol. CXXVI, p. 424, 1931.
Comberg.—*Ber. d. deutsch. Ophthal. Gesellsch.*, Vol. XLVIII, p. 322, 1931.
Fisher, J. H.—Zentrabl. f. Ophthal., Vol. XXVI, p. 811, 1931.
Fischer, F. P.—*Klin. Monatsbl f. Augenheilk.*, Vol. LXXXVII, p. 537, 1931.
Gonin.—*Ber. d. deutsch. Ophthal. Gesellsch.*, Vol. XLVII, p. 46, 1929.
Guist.—*Ber. d. deutsch. Ophthal. Gesellsch.*, Vol XLVIII, p. 343, 1931.
Imre.—Ber. d. deutsch. Ophthal. Gesellsch., Vol. XLVIII, p. 321, 1931.
Klein.—Klin. Monatsbl. f. Augenheilk., Vol. LXXXVII, p. 537, 1931.
Lindner.—*Arch. f Ophthal.*, Vol. CXXIII, p. 233, 1930.
Luntz.—*Zeitschr. f. Augenheilk.*, Vol. LXXIII, p 380, 1931.
Majewski.—*Zentralbl. f. Ophthal, Vol. XXIV, p. 637, 1932.*
Weve.—*Klin. Monatsbl. f. Augenheilk.*, Vol. LXXXVII, p 145, 1931.

A CASE OF CONGENITAL CATARACT SHOWING UNUSUAL FEATURES

BY

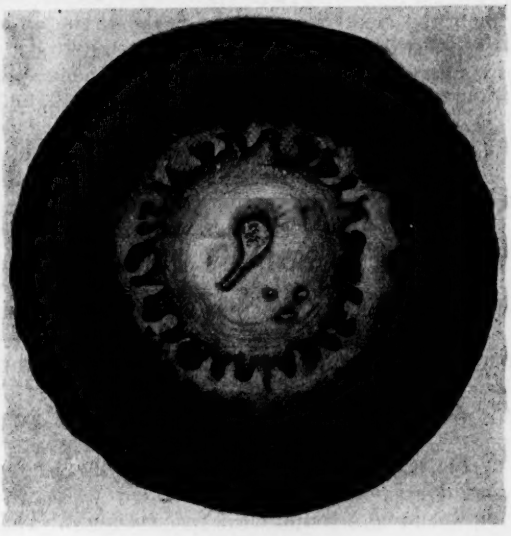
D. J. WOOD

CAPE TOWN

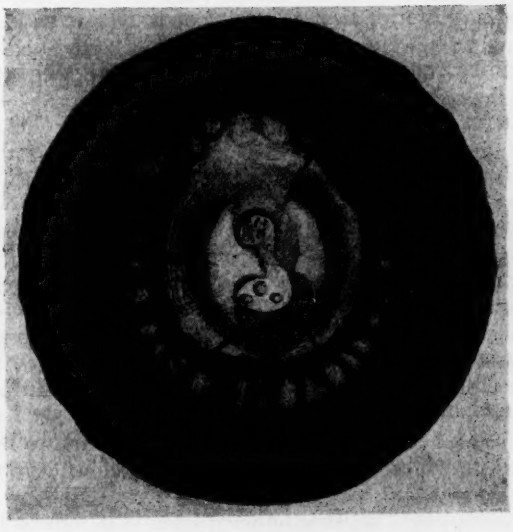
A HEALTHY and well grown Swahili boy was brought to me from the Athlone School for coloured children. There was nystagmus, and a dense white opacity in each pupil which reduced his vision to mere light and dark.

The iris acted normally, but on dilating the pupils a very remarkable appearance presented itself which I have tried to depict. The drawing was difficult and tedious owing to the nystagmus, the nervousness of the patient, and my inability to speak his language. The lights and shadows are exaggerated by the brilliant light of the slit lamp, and I have taken some liberties with the irides as they were normal and not associated with the actual abnormalities.

In the centre in each eye was a very dense circle of capsular opacity, with a yellowish raised epicapsular spot, and several smaller specks, remains of lens matter. These were more numerous in the



LEFT EYE



RIGHT EYE

left eye. The central dense part was marked by concentric lines, rather bluish in the right eye, and giving the tissue an appearance reminiscent of tendon.

The curious part was the presence round this central opacity of a flat slate-coloured band, notched at its inner margin, and furrowed on its surface. The markings were so dark that at first one thought them due to actual pigment, but more careful examination showed that they were thinner parts through which the dark interior was dimly seen. The appearance in the right eye indicated that the markings were due to the radial ridges of the iris uvea. The left was more irregular but also suggestive.

It was evident that the lens was largely absent and was represented only by capsule, but why the centre was dense, and the periphery thin and stamped by the impress of the uvea was not clear. The general appearance was similar in the two eyes, and postulated a common cause.

As there was a definite border between the dense central part and the thinner outside band I attempted to separate them with a discission needle, and found this quite easy, so that one left a round disc of capsule loose in the anterior chamber. This was removed the following week and a round clear space was left. Thereafter the drawing of the left eye was made and the same treatment followed.

The round portion of the capsule, 5 mm. in diameter, was examined under the microscope and showed an anterior surface marked by digitate wrinkles outlined by very fine round pigment atoms, and a posterior surface with concentric wrinkles, and oval nuclei lying in lines corresponding to them. No nuclei belonging to the anterior capsule were visible. What were seen appeared to represent the only remains of the lens. They stained feebly.

The fundus was normal and the vitreous clear. Though there was uveal pigment in very fine particles on the anterior surface of the capsule there was no trace of iritis.

The boy sees well with his glasses but so far is illiterate.

Many years ago I saw a patient who had been struck on his eye by a non-penetrating pellet. His lens presented a condition faintly like the present case. The uveal marking of the iris and of the edge of his pupil were indicated by lines resembling what might be made by a soft lead pencil. It was, however, only a ghost compared with the present case.

It is clear that the cause of the trouble is prenatal, but it is not so clear at what time it occurred nor what was the mechanism.

The epicapsular deposits seem to indicate that the lens capsule had been opened in each eye, but that there had been no intra-uterine iritis seems also certain.

May one suppose that the thinner part of the lens capsule in the centre was so far abnormal that it gave way as the lens grew, and

allowed the aqueous to penetrate and ultimately dissolve the whole lens substance? The swelling of the lens during the earlier stages might bring the lens capsule against the iris, with the result that the pattern of its uvea remained as a permanent appearance. It seems a possible explanation.

A CLINICAL NOTE ON GLAUCOMA

BY

E. E. MADDOX

BOURNEMOUTH

THE following simple addition to our ordinary measures for reducing the tension in most cases of glaucoma seems worth communicating.

About five years ago I was much interested to discover that high frequency currents had the effect of almost immediately lowering the tension in nearly all *moderately* hypertense eyes.

In one case only during these years has an untoward sequel resulted. The patient had toxic cyclitis with secondary glaucoma, and the treatment was followed by a temporary increase of hardness and pain. It was this case that made me delay publication till further experience should prove the method safe enough to recommend. Caution, therefore, should be observed with inflamed eyes. With very hard and strangulated eyes the effect may be nil.

The application need not be longer than from one to four minutes, and although the effect is more or less transitory, I find that in most cases, repeated applications do good in the long run. This indeed is what we might expect, for a temporary reduction of tension must allow the oppressed structures a breathing space in which to recover themselves from the continuous pressure, thus allowing a flushing with fresh blood and consequent increase of nourishment.

Until recently I have applied the current, either directly or through the operator's fingers, to the eyeball itself through the closed lids; but now find it makes little difference if the application is made instead against the temporal margin of the orbit. In order not to startle the patient, the current should not be turned on until contact has been made with the skin, as otherwise slight sparking is caused. In any case the patient may be assured that there will be no shock.

The lowering of the general blood pressure by ordinary high frequency currents applied to the body has long been a matter of common knowledge, but the local effect of these weak currents on glaucoma does not appear to have attracted attention, and will, I hope, prove a useful addition to our ophthalmic armamentarium.

A SERIES OF ONE HUNDRED CASES OF HYPOPYON ULCER, WITH SPECIAL REGARD TO A METHOD OF TREATMENT

BY

A. CHRISTIE REID

HON. SURGEON, NOTTINGHAM EYE INFIRMARY

ALL will agree that it is of primary importance to arrest at the early stage any infected ulcer of the cornea. In industrial practice one sees many such cases at a stage where hypopyon has not appeared. These may have a considerable infiltration area and even sometimes a posterior abscess of the cornea, as proved by Mr. B. Samuels in his recent paper.[1] My method of treating these, of which I have had a considerable number in the period under review, is the same as that elaborated for the more severe type with a developed hypopyon, *viz.*, prompt application of a modified Wessely cautery, with or without rest in bed. Such cases are not included in my series. They have given with this method 100 per cent. of successes, *i.e.*, recovery with vision 6/12 or more. One or two out of several score have developed a minute hypopyon on the day following, but this has rapidly cleared.

This series comprises cases only which when first seen by me had a hypopyon visible to the naked eye.

In a previous communication[2] I adopted a rough but practical classification of results in another, earlier series, also of 100 cases, thus:—

(*a*) Good, with vision 6/6 to 6/18, or very slight scar.
(*b*) Fair, 6/18 partly to 6/60, or with denser scar.
(*c*) Bad, less than 6/60 or very dense scar.
(*d*) Loss of eye.

The vision alone is of course not always a criterion, *e.g.*, in nystagmus cases (most of my cases are miners), refractive error, amblyopia, etc., and in these the degree of scarring must form a basis of classification. One arrives by experience at a position in which one can say with some confidence "This is a scar that should allow 6/18 or only 6/60, or even less." Not half-a-dozen cases, which gave a visual result altogether out of proportion to the amount of scarring, have been so dealt with, *i.e.*, moved into a class one higher in the scale. One moderate application of the cautery referred to does not cause dense scarring.

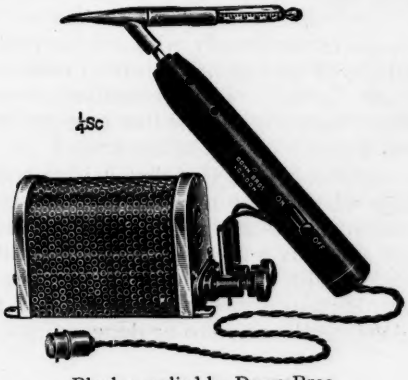
The question of bacteriological diagnosis need not detain us. We take for granted that the pneumococcus is the causal organism in the vast majority and that the tendency is for its spread to be along the surface, undermining the epithelium which rapidly breaks down. The spread occurs mainly, I think, outwards in all direc-

tions equally from a centre; and does not penetrate more deeply than 1/4 of the corneal thickness, in the stage at which one usually first meets with the ulcer. Thus the deep abscess need not be attacked, as it is sterile like the hypopyon itself. A few cases may be due to an organism of the diplobacillary group. In one of my earlier series I found one of the Petit type. In one of this series, No. 75, which showed chronic blepharitis of diplobacillary type perforation occurred. Perhaps these cases are more liable to perforate.

In my former series I argued for an early section of the cornea by a special incision which I described as likely to avoid iris prolapse, but in this new series I abandoned early section and reserved section for later cases that had done badly, usually with methods other than my cautery. Early section may be efficient in very many cases but it is a more severe method and (one grave objection) it does not allow of prompt dealing with an infected tear sac. It is too dangerous to open an eye within, I think, three days of excision of a sac. This objection does not hold in the case of the mild cauterization I am recommending in this paper.

I have come to the conclusion that the safest and most satisfactory method of treating all hypopyon ulcers at whatever stage they present themselves is one or more applications of a cautery of the Wessely type along with certain adjuvant methods which I will refer to later.

I found the German instruments very liable to get out of order and they could not be set right again without the delay involved in sending them back to Germany. Messrs. Down Bros. of St. Thomas's Street, S.E.1 have made some very practical improvements in the instrument and have given it the name Metri-cautery, in view of the very fine regulating mechanism, that allows of application at an exact temperature at the point, of between 70° and 90° C. A thermometer reaching almost to the tip registers this. 70° is



Block supplied by Down Bros.

said to be fatal to the pneumococcus. 90° is apt to do more damage to the cornea than is necessary. I advise, therefore, 75° to 85° as the optimum. A few seconds suffices for the point to be stroked over the surface, carefully exploring the edge where advance seems likely, and even breaking through into any fresh focus, without, as I said, attacking the secondary deep infiltration abscess, if present. I have abandoned the red hot cautery. This, used even with the care of Mr. Edgar Stevenson's[3] method of approach, *i.e.*, not actually in contact, has in my experience produced such dense scars and has led so often to perforation that I have come to regard it as too dangerous and damaging. This may be because the red and infra-red rays penetrate more and cause a deeper reaction than is necessary. My cautery does not act in virtue of rays. It is in contact the whole time of its application.

Every series will include cases brought too late for any method to offer any hope of success. Seven were classified as such and of these three were women. Women put up with things longer than men, and often come too late. Out of a total of seven females, two were so bad on admission that the eye was lost, and in two others the result was "bad."

These four female cases may be analysed thus:—

No. 98 came with a three weeks' history of an inflamed eye and the whole cornea on admission was deeply infiltrated with pus.

No. 97 with a 10 days' history, general condition poor, aged 69 years.

No. 92 also aged 69 years, a syphilitic with iritis and old standing chorio-retinitis, left with a dense scar.

No. 96 a 10 days' history. My house surgeon did a corneal section in my absence and as this was not checking the advance of the ulcer, I cauterized it two days later and arrested it, but vision was only 2/60. I do not recommend this sequence.

Cases 98 and 97 lost the eye.

But taking the severe and even hopeless cases along with the less severe and even mild cases, the series shows "good" results in 65. This compares favourably with the previous series which by various methods of treatment, of which this cautery was not one, gave only 48 "good." Further analysis is required. How many cases were of such a mild type that it might be said that any simple treatment would probably be a success?

I would here emphasise the fact that it is impossible to say at the earliest stage whether the ulcer will assume a virulent form or not. The case regarded as diplobacillary came at first with no history of injury and only a tiny subcentral ulcer. He was treated by being sent home with zinc lotion and atropine. He turned up a week later with his anterior chamber half-full of pus, eventually perforated, and evisceration had to be done.

Previous sad experience has brought home to me that every case coming within 24 hours of an injury with an ulcer that has any appearance of being infected should be seen again the next day, if, indeed, it cannot be admitted straight away to the ward.

There were in this series 18 cases of so mild a degree that methods other than cautery proved successful. They presented themselves either before we got the cautery, or during a period when it was out of action, or in my absence before I made it a routine treatment.

The results were "good" 17; "fair" 1. This last was in very poor health, was iritic, and insisted on home treatment.

In only two of these cases was the duration stated at over five days, in eleven it was less than five days, in the remaining five it was not stated. Two of these last were iritic, and it is difficult to be accurate about the date of onset.

But experience teaches that even these mild cases should be treated with respect. I insist now on every case being put to bed, laid on the back, allowed only one pillow, getting up only for needful calls, meals, etc., the idea being to diffuse the pus over a wider absorptive area. The patient is allowed a bowl of perchloride of mercury 1 in 10,000 beside him, and is instructed to bathe the eye freely every half hour during the day, and as often at night as he wishes. Argyrol 20 per cent. drops are instilled by a nurse every 2 hours. This is kept up for 48 hours. If then there is no advance, cautious sitting-up in bed is allowed, followed by getting up in the ward in another 48 hours; and two days after, if no advance, and the surface taking the argyrol stain all over, the patient is sent home. The most favourable cases thus demand six days in hospital.

In this series, as I said, various forms of treatment were given, and the application of *pure carbolic* which used to have a place with me, was one. 18 cases were treated by free swabbing with carbolic. In 11 of these it proved inadequate, and other methods had to be substituted. In fact I considered that valuable time was lost. I have given it up.

The red hot cautery, not touching, was applied in five cases. The results were very disappointing, *viz*: "good" 1; "fair" 2; "bad" 2. The good one perforated, but one cannot regard this as a triumph. In my former series, of a small group no fewer than six perforated. I regard the rays as dangerous.

Corneal section was reserved for the worst cases, after other methods had failed. In only two was this method adopted after the Wessely cautery. In one of these, No. 23, my H. S. applied the cautery and on the following day, as the ulcer did not seem to be arrested, I sectioned the cornea. The eye went from bad to worse and had to be removed. I had at that time not a sufficient

faith in the instrument. I should have persevered with it, even to several applications if necessary. The other case, No. 97, was that of an old woman, aged 69 years, with a ten days' history, in whom three applications proved inadequate and as a last hope I sectioned the cornea. The eye had to be sacrificed. Three valuable days were lost before the cautery was applied, owing to my absence. If the case had been attacked with vigour on admission the result would, I feel sure, have been more favourable.

The results of section done under these conditions were: "fair" 6; "bad" 4; "eye lost" 5, total 15.

One learns more sometimes from failures than from successes. An analysis of the ages of 11 cases that resulted in "loss of eye" shows that 11 of these were over 60 years and only 3 under 50 years. Poor general condition, tear sac infection, septic teeth and of course the duration of the ulcer weigh heavily against success. One was a thorn injury with severe iritis on the second day. Thorns seem to contain some very irritating substance. In 4 of these 11 was the Wessely cautery tried:—

No. 23 above referred to.

No. 33, with a 10 days' history, continued at work and came with cornea necrotic, and anterior chamber half full of pus, the eye perforated.

No. 75, already referred to as possible diplobacillary, lost a week of valuable time on inefficient home treatment. After two cautery applications, free swabbing with 2 per cent. zinc sulphate was tried but proved ineffectual.

No. 97 is referred to under corneal section.

In three of these lost cases then the cautery was applied too late, *viz.*, after the tenth day. In the remaining one it was applied half-heartedly after the fourth day and I think wrongly followed by section.

Turning finally to the total group of cases treated by the Wessely, we have the following encouraging results:—"good" 31 or 68·8 per cent.; "fair" 9; "bad" 1; "loss of eye" 4, total 45, of which only 4 after seventh day:—resulting in "fair" 1; "bad" 1; "loss of eye" 2, showing again the importance of early effective treatment.

In 38 the Wessely was the only special treatment. In two iritic cases intravenous Sodii Salicyl. was also given. In five the eye was sectioned, in only one of which was the result "fair," the others being "bad" or "loss of eye."

Comparing now the over-all results in my two series, we have:—

First Series. "Good" 48; "fair" 13; "bad" 30; "loss of eye" 9=100.

Second Series. "Good" 65; "fair" 16; "bad" 8; "loss of eye" 11=100.

And when the Wessely treatment alone is taken, the figures are even better, as above.

I fear we shall aways be left with say 10 per cent. of cases that will be too far gone to expect a good result with any method.

REFERENCES

1. *Trans. Ophthal. Soc. U.K.*, Vol. LI, p. 485.
2. *Lancet*, 1929.
3. *Trans. Ophthal. Soc. U.K.*, Vol. XLVII, p. 55.

MARKED PAPILLOEDEMA IN PULMONARY EMPHYSEMA

BY

ALISTAIR J. CAMERON, M.B., CH.B.Edin., D.O.M.S.(Lond.)

CLINICAL ASSISTANT, OPHTHALMIC DEPARTMENT, ROYAL INFIRMARY, EDINBURGH

THE following case appears to be unique and consequently may be of interest.

P. B., a male, aged 34 years, a coalminer, was admitted to the medical wards of the Royal Infirmary, Edinburgh, on October 26, 1932, under the care of Professor Murray Lyon, to whom my thanks are due for access to the notes on the general condition.

His complaints were:—

(1) "Shortness of breath of two years' duration."

(2) "Defective vision in both eyes of two months' duration."

On examination by Professor Murray Lyon the case was diagnosed as one of extreme emphysema, unusual in the fact that the cyanosis and congestion were confined to the upper part of the body.

Examination on admission showed:—

A well-developed deeply cyanosed man, lips and ears dull purple; centre part of the face deep bluish red; breathing shallow and apparently difficult; eyes prominent and congested; fingers clubbed.

Blood pressure normal, 130 mm. Hg systolic, 85 mm. Hg diastolic. Heart: sounds closed.

Alimentary system: no history of vomiting at any time.

Spleen not enlarged.

Central Nervous System: no loss of motor or sensory power.

Reflexes: pupils equal and active to light, direct and consensual, and to accommodation. Other reflexes showed no abnormality.

Cranial Nerves:

(1) Subjectively unimpaired.

(2) Mistiness, dimness and, occasionally, vision only for movement.

(3) Slight ptosis of left eyelid dating from infancy.

(4) No nystagmus.

(6) No nystagmus.

(7) Nothing to note.

(8) Nothing to note.

(12) Nothing to note.

No headache. Wassermann Reaction negative.

Careful repeated examination failed to reveal any disease of nervous system.

Urine: nothing abnormal.

Dark Room examination revealed: intense venous congestion of both retinae, all the veins being bulging, tortuous, definitely plum-coloured and several times the calibre of the arteries; some small flame-shaped haemorrhages were seen in the region of the discs, particularly in the left eye. In each eye there was an extreme degree of papilloedema of the hob-nailed variety with steep banked edges and with plum-coloured congested veins.

On direct examination the swelling in the right nerve head measured 5 D., in the left nerve head 6 D. Right vision 6/6 and J. 1. Left vision 6/12 and J. 2.

The field in each eye was not contracted on examination with medium-sized objects.

On examination on November 3, 1932, the patient was found sitting up in bed and reading. The swelling of the discs appeared to be a little less marked.

On December 1, 1932, the patient's general condition was so far improved that he was examined again prior to discharge. The few retinal haemorrhages had by this time completely disappeared. The distension of the veins was still marked and the tortuosity more pronounced. The swelling of the discs was not so congestive. The right papilloedema measured 4 D.; the left about 5 D.

R. V.=6/9 part, J. 2; L. V.=6/60.

The field in the right eye on examination at the perimeter with a 10 mm. object was practically full with a slope into the 30° circle in the lower nasal quadrant but the field in the left eye was concentrically contracted down to about the 15° circle with a 20 mm. object. The consecutive atrophy appears likely to lead to blindness.

The patient's chest condition and his general condition are now much better. He has not at any time, although under continual observation, shown any signs of any intracranial disease.

As far as I have been able to review the literature I have not come across a similar condition.

Mackenzie[1] refers to a case with great tortuosity of the retinal veins in a man the subject of emphysema but there is no reference to swelling of the optic disc.

Pick[2] mentions a case of emphysema with great dyspnoea and retinal haemorrhages in both eyes.

I am indebted to Dr. Traquair for checking the measurements of the swelling of the discs and for his courtesy in permitting me to publish this case.

REFERENCES

1. **Mackenzie, Stephen.**—*Medical Times and Gazette*, Vol. II, p. 765, *December 23, 1882.*
2. **Pick.**—*Arch. f. Augenheilk.*, Vol. XXXIX, S. 382.
3. **Groenouw.**—*Graefe-Saemisch Handbuch*, Dritte Auflage, Abt. IA, S. 26.

ANNOTATION

The Certification of Blindness

A paper under the above title is contributed to the *Medical Officer* of December 24, 1932, by N. Bishop Harman. In it he refers to a previous paper by Dr. G. W. N. Joseph in the issue of October 29, which deals with the report of the Union of Counties Association for the Blind, in which the author demands (1) a single definition of blindness; (2) a uniform certificate; for which purpose he gives his support enthusiastically to the form devised by the Union of Counties Association. We have not seen Dr. Joseph's paper, but any remarks by Bishop Harman must of necessity hold the attention of all ophthalmic surgeons. Bishop Harman calls attention to the steady increase shown in the numbers of the blind year by year since provisions for the blind have been devised by legislation; and this in spite of the success obtained in the diminution of blindness in children from ophthalmia neonatorum. This large increase is in the number of the blind in the later years of life, and it is likely that the increase is due to increasing disclosure of cases rather than to any additional production of blindness. It is now worth while for a blind person to disclose his disability, and we should be the last to complain that this is so. But it is only reasonable to demand some assurance that only the genuine economically blind are receiving benefits.

A single definition of blindness from the point of view of the legislature is impossible; for certification is required under three separate Acts of Parliament, namely, Education, Blind Persons and Old Age Pensions. From the education point of view blindness means that a child is unable to see to read the ordinary school books. For the Blind Persons and Old Age Pensions Acts blindness means "so blind as to be unable to perform work for which eyesight is essential." The wording differs, but in effect they have the same meaning, since for the child the reading of books is its work.

No "yard-stick, measure or formula, which shall include all the relevant factors of sight set out in plain figures" is possible. Bishop Harman gives brief details of three cases: the first, a chronic glaucoma with no perception of light in one eye has, with correction, a central acuity of 6/9 in the other and a field limited to the 5° circle. Her acuity enables her to read *when she can find the place*, but she has to be led about. The second case is that of an old parson with senile central choroiditis, an absolute central scotoma and a central acuity of 0, with full peripheral fields. He can do full duty as he knows the prayer-book by heart, but he cannot see the face of a parishioner. The third case is that of a city man of 37 years of age with retinitis pigmentosa. His central acuity is 6/5; he has full fields in a good light but is severely handicapped in reduced lighting.

No universal formula will serve these cases, but only the statutory reference to work.

In the case of children Bishop Harman points out that the certification of blindness can only rarely be final; it should always be regarded as provisional and subject to revision. Where no full provision for children with defective sight exists it is inevitable that border-line cases will be drafted into the blind schools. The cure for this state of affairs is establishment of sight-saving classes. With regard to the form of certificate, Bishop Harman, while paying tribute to that devised by the Union of Counties Association, urges some criticisms of it which appear to be pertinent. He points to its complexity, including as it does 187 spaces, and notes that no space is provided for *diagnosis*. He contrasts this elaborate form with the simplicity of Form R.M.14 of the Ministry of Health for "Appeal cases—Blindness," which only has four spaces; a line for diagnosis, a space for the facts of the case, a space for IS or IS NOT in the declaration of blindness and a space in which the certifier is to give his reasons. He does not think that elaboration increases the value of the information given. He regards the form as dangerous in that "hearsay" is ranked alongside the facts ascertained by the certifier as though both were of equal value; *e.g.*, in section 3, c. (1) (*a*) and (2) (*a*) and (*b*). The ophthalmic surgeon is directed to "state precisely" or "state precise cause" of happenings or events which are outside his ken. The reference is to accidental causes of blindness. The actual certificate is also faulty. Section (2) relating to school children is wrong in its implication. Twice the word TOTALLY (in heavy-faced type) is used to qualify the word blind. It is true that there is a saving clause, but the use of the word "totally" is irrelevant and wholly wrong, for it is contrary to the definition of the Education Act and does not apply to the majority of children who are properly pupils of blind schools. There are other points in Bishop Harman's

interesting paper to which we should have liked to draw attention, but as we append his essentials of a model form of certificate, we feel that we have overstepped the customary limits of an annotation.

"*The Essentials of a Model Form.*

To my mind a model form must be short. It must be designed to secure the maximum of matters of fact and a minimum of hearsay. Each question must be self-contained, and there must be no footnotes. I submit such a form. It is for the most part one in use by the largest statutory authority in the country. It works well. I have added a list of causes of blindness so that there may be a common usage of terms. The list is the shortest I can devise with the idea of prevention in view. I believe it will enable us to get all the information that we can expect to get. We are not, or ought not to be, aiming to get the maximum of *interesting* data but the maximum of *useful* data. This list would get it. A score of thousands of returns made by approved practitioners on this plan would be of real value.

CERTIFICATE RELATING TO BLINDNESS.

I hereby certify that I have examined.....
..... aged..... .....of..... ....
and find that his vision is as follows :—
her

Without glasses :— With glasses :—

R.V.
L.V.

Field of vision (when acuity is above 3/60) :
(*a*) Full.
(*b*) Moderate contraction.
(*c*) Marked contraction.
(*Please strike out definition not appropriate.*)

NATURE OF VISUAL DEFECTS.....
.....
.....
.....
.....

DIAGNOSIS (Use terms in list printed on back of form).....
.....

(1) I am of opinion that.....is.....
so blind as to be unable to perform any work for which eyesight is essential within the meaning of the blind Persons Act, 1920. (*Note :* this disability refers to *any* work and not to his or her own occupation ; and it must be distinct from any other disability of mind or body).

(2) If the person is a child under 16.
(*a*) Is it blind within the meaning of section 69 of the Education Act, 1921, namely, "too blind to be able to read the ordinary school books used by children," so that it can only be appropriately taught in a special school or class for blind children ? (The answer should be Yes or No).....
(*b*) Has the child such bad vision that it cannot read ordinary school books without risk of injury to its eyesight or that it cannot with advantage be taught in an elementary school and should therefore be taught in a Special School or Class such as a myope or sight-saving class ? The answer should be Yes or No).....

PROGNOSIS. (To be answered if possible, Yes or No)

(3) If the person is a child under 16, do you consider it likely that the child will be blind within the meaning of the Blind Persons Act, 1920, on attaining the age of 16 ?.....

(4) If the person is 16 years of age or over and not at present blind within the meaning of the Blind Persons Act, 1920, do you consider it likely that the person will become blind within the meaning of the Act during the next 4 years ?.....

Date.....

Signed.....

(Registered Medical Practitioner.)

Qualifications.....

Address.....

CATEGORIES OF CAUSES OF BLINDNESS.

Congenital defects or disorders.
Myopia and sequelæ.
Inflammatory diseases and sequelæ :—
- Ophthalmia neonatorum.*
- Purulent conjunctivitis of later years.*
- Trachoma.
- Superficial keratitis.
- Interstitial keratitis.*
- Iritis and irido-cyclitis.*
- Choroiditis.*
- Optic neuritis.*

Tumours or malignant disease :—
- Ocular.
- Intra-cranial.*

Degenerations and vascular diseases :—
- Retinitis.
- Retinitis pigmentosa.
- Central senile choroiditis.
- Glaucoma.
- Cataract.

Accidents or toxic effects, and their sequelæ :—
- Casual.
- Industrial.
- War.

Cause unknown.

* (If possible note whether due to V.D. or not.)"

INTERNATIONAL ASSOCIATION FOR THE PREVENTION OF BLINDNESS

Annual Meeting, Paris, November 19, 1932

THE Annual Meeting of the International Association for Prevention of Blindness was held in Paris, 66, Boulevard Saint-Michel, on Saturday, November 19, 1932, at 2.0 p.m., under the Chairmanship of Professor de Lapersonne and in the presence of Monsieur Justin Godart, Minister of Public Health. Monsieur de Monzie, Minister of National Education, was represented by his First Secretary, Monsieur Abraham. H.R.H. the Duchess of Vendôme was represented by Mademoiselle Szymanska de Slepovron.

The PRESIDENT presented the Annual Report, showing the regular progress achieved by the International Association and its French Committee since their formation. He thanked the National Society for the Prevention of Blindness in the United States, which had, as previously, doubled all subscriptions collected in Europe

and he begged Mrs. Hathaway, Associate Director of the National Society, who had undertaken a long journey in order to attend this meeting, to express to her colleagues, his earnest gratitude.

Finally, he reminded his audience that the present meeting was devoted to the creation of sight-saving classes and he mentioned the names of the eminent experts who had kindly consented to take part in the discussion: Mr. Bishop Harman, the founder of sight-saving classes, Mrs. Winifred Hathaway, of the United States, Professor Bartels (Dortmund), Dr. Dufour (Switzerland), Dr. Redslob (Strasburg).

Professor VILLEY explained eloquently the reasons for which the adoption is necessary of special educational methods for children who do not see well enough to follow ordinary classes yet see too well to be sent to institutions for the blind. He declared that while a great deal has been done for the blind child in most countries, very little has been done for partially seeing children.

Dr. HUMBERT, General Secretary of the Association, then read the report of Mr. BISHOP HARMAN, who was prevented from attending the meeting. Mr. Bishop Harman stated that it was his duty of inspecting children in schools for the blind, maintained by the London Education Authorities, which first brought home to him the difficulties of these children who are neither blind nor well-sighted and for whose education and training there was no appropriate provision. He found that no less than 6 per cent. of the children in these blind schools were myopes but were not blind and would probably never become blind.

The first sight-saving class was created in London in 1908. To-day there are in London 37 classes in 15 centres with a roll of 180 children. There are also 100 places for myopes in five schools for higher education.

An examination of the records of a large number of children who have been in the classes shows that they fall into three groups:—

Myopia	62·62 per cent.
Damage done by inflammation ...	30·35 per cent.
Congenital defects	7·05 per cent.

The author laid stress on the aggravation of myopia during the school period. At first the standard for children admitted to these classes was set at 6/18 with glasses, but it was soon found that many with worse vision than this could satisfactorily join in the classes; when the vision is less than 6/24 it is better to exclude these children from sight-saving classes.

A child, of 5 years with 5 D. of myopia, especially if there is an inclination to increase of the defect, is certainly in need of special treatment. On the other hand, a child with 7 D. who is 13 years old, and is shortly due to leave school, may well be left in the ordinary school, under special supervision and exemptions as regards

work. At the outset these classes were affiliated with the ordinary schools and, as far as possible, the children in the classes took the same oral teaching with the normal children. Gradually under the influence of the teaching profession and modern educational methods, these classes became completely separate. There is a tendency, however, for economic motives, to return to the original system.

The author thinks that the real proportion is not less than one short-sighted child for every five-hundred school children. Estimates from the United States agree with these figures.

Mr. Bishop Harman advocated mainly oral teaching and manual work; he was not in favour of the publication of special books for these children, as the medical aim was to secure a good education for the children with the least risk, and at the same time, to cultivate a life habit of *not* reading.

Among the occupations for the partially sighted he advocated pharmacy, housework, farming, nursing, massage, gardening, etc.

Mrs. WINIFRED HATHAWAY, Associate Director of the National Society for Prevention of Blindness in the United States, mentioned that it was Edward A. Allen, Director of the Perkins Institute for the Blind, who first conceived the idea of instituting special educational methods for the partially sighted in his country.

The first sight-saving class in America was created in Boston in April, 1913. To-day there are 409 sight-saving classes in the United States representing 118 cities and 22 States.

Following a meeting at the University of Chicago of oculists and education authorities, the following rule for the selection of children to be sent to sight-saving classes was adopted.

Children with a visual acuity of 20/70 (6/21) or less in the better eye after proper refraction.

(*a*) Children in elementary schools with four or more dioptres of myopia.

(*b*) Inactive, subsiding (or regressive) cases of interstitial or phlyctenular keratitis, optic neuritis, trachoma, etc.

Children with a visual acuity of 6/60 will not in all probability be able to use sight-saving class equipment and are recommended for Braille work. The proportion of children requiring the advantages of sight-saving classes is 1 to 500 or 1,000 school children.

The author was distinctly in favour of mixing up as far as possible the short-sighted children with their normally sighted school-fellows.

According to the system in use in 90 per cent. of these schools in the United States, these children follow separate classes for all work necessitating close use of the eyes. They join the normally sighted children for all other lessons such as singing, music, acting and other activities of the common programme.

Mrs. Hathaway insisted on the proper lighting, decoration and furnishing of the school-room. Artificial lighting must meet the

same requirements as natural lighting, sufficient illumination for the type of work to be accomplished, well diffused, well distributed and without glare.

The subjects taught in the special classes include reading, writing arithmetic, handiwork and typewriting. The so-called social subjects—geography, history, natural science, appreciation of music and singing are taught in the regular classes in common with the normally sighted children, but the preparation for these subjects is done by the sight-saving class teacher.

Approximately 4·5 per cent. of the pupils of sight-saving classes can, later on, go back to the ordinary classes.

In some districts where the transport facilities are good, rural sight-saving classes have been established.

A few attempts have been made in view of the vocational training of these cases, but much remains to be done in this line.

In America 14 States have appropriated out of their school budget a special subvention for sight-saving classes.

Mrs. Hathaway concluded by saying that the aim to be achieved was not so much to multiply these classes as to render special education unnecessary through the reduction of the number of partially sighted children by proper methods of sight preservation.

Professor BARTELS (Dortmund) explained the motives which made special educational methods indispensable for partially sighted children. He considered that only a myope whose sight between 6 to 8 years of age was −8 D. must attend a sight-saving class. Children with a lower degree of myopia are only exceptionally admitted to these classes. He observed that in Anglo-Saxon countries myopes with −4 D. are sent to sight-saving classes.

He agreed with previous speakers that only mentally normal children may be admitted to these classes. He also agreed with them in estimating the rate of these children at one to two per thousand school children. This figure, he stated, was much higher if one admitted children with −4 D. of myopia.

At a Congress of teachers in blind schools in Germany it was stated that the number of children brought up in these schools who ought to attend sight-saving classes had been estimated at one-fifth.

The detection of children with weak eyesight takes place in Germany by means of medical school inspection. Teachers in the primary schools must examine the children periodically according to a set formulary, as many children become shortsighted during the school period. Children with defective eyesight pointed out by the teacher are submitted to an examination by an ophthalmologist. So far it has been noted that approximately 10 per cent. of these children are candidates for the sight-saving class. In the country the detection and education of partially sighted children are beset with great difficulties.

The author mentioned three methods of education for these children.

1. Independent sight-saving schools.
2. Sight-saving schools annexed to institutes for the blind.
3. Separate sight-saving classes in ordinary schools.

The last method has the advantage of bringing together partially sighted and normal children. However, the author is of opinion that only proper sight-saving schools permit an accurate adaptation of educational methods to the sight of each child so as to obtain the maximum result from such teaching.

In Berlin there are at present three sight-saving schools including 350 pupils and 24 classes.

The special school in Dortmund has received 58 children and includes five classes. Of the 25 children who have finished their classes, five have returned to the ordinary school and two have been transferred to a higher class.

Educational expenses, although rather higher in sight-saving than in ordinary schools, are however, far below the cost of teaching a blind child.

In Germany, teachers in sight-saving classes are expected to follow a post-graduate course of one year at the Pedagogic Institute in Berlin. The author did not think it is possible to get good teachers for this special form of education with the six-weeks' course which is adopted in other countries.

From the educational standpoint, the author described with great detail, the manner in which these children are taught to read and write. One method he said, was with large characters of 10 centimetres cut out of cardboard and covered with cotton stuff which the child learns to distinguish by touch. Later, one uses letters in yellow material on a blue background or black on yellow, the capital letters being 10 centimetres in length and small letters 6/7 centimetres. Gradually the child is taught to recognize the smaller letters. The only reading books for short-sighted children in Germany are the four books of the special schools in Hamburg. The author mentioned that reading and writing presented special difficulties for the partially sighted in Germany on account of the fact that these children have to learn not only the Latin but also the Gothic letters.

In Germany, as everywhere else, one has attempted to train these children in manual work. This training is beset with many obstacles and in some cases the only resource is to send these children to a vocational school for the blind.

Dr. A. DUFOUR (Lausanne) stated that it was in the spring of 1925 that the first sight-saving class was opened in Switzerland at Zürich. The second one was started in 1930 at Bâle.

In both these cities special teaching is carried out according to

the following principles, which have been adopted since 1918 by the Central Swiss Union for the Welfare of the Blind.

(*a*) Sight-saving classes admit children from 6 to 14 years of age, whose visual acuity in the better eye after correction, is below 0·2 (Bâle has admitted 0·5).

(*b*) These children are admitted with the consent of their parents on the recommendation of the teacher, the school medical inspector and an ophthalmologist.

(*c*) The classes, which must not exceed 20 pupils, are periodically visited by an ophthalmologist.

(*d*) The teaching methods are adapted to the degree of development of the child and are therefore quite individual, attempting to spare his weak eyesight by being chiefly oral. While developing the senses and organs one endeavours to strengthen the physical, intellectual and moral condition.

The benches in the schoolrooms have backs, the desks are adjustable and each carries its own blackboard. Books printed in thick letters are used and a magnifying glass is employed when reading from the books of the ordinary school.

Dr. REDSLOB (Strasburg) stated that one of the earliest sight-saving classes had been created in 1911 at Strasburg. The Municipality maintains these schools, it even supports the expenses of tramway fares for children who need to be taken from Strasburg to St. Thomas, where the school is situated. The selection of children for admission to the school is made in the following way:

A first selection is made by the medical inspector of the municipal schools, who examines every year the eyesight of school children.

Those with very defective eyesight are referred to Dr. Redslob, Ophthalmologist and Medical Inspector of the municipal schools, who has assumed this task since the school was created and decides, from a medical point of view, whether the child should be admitted. As a basis, Dr. Redslob assumes that a pupil whose visual acuity in the better eye is equal or inferior to 2/10 should be admitted to a sight-saving class with certain individual exceptions according to the result of the medical examination. These figures have been adopted in other countries which have followed the lead given by Strasburg. On the other hand, children whose better eye only perceives fingers at a distance of 1 metre are sent to an institute for the blind.

Since the creation of this special school one has been able to withdraw from the Institute for Blind Children a number of pupils who have been sent to the sight-saving classes, greatly to their advantage.

The meeting was followed by the projection of a documentary film on sight preservation among children and an exhibition of schoolroom material brought from England by Miss Young, from the United States by Mrs. Hathaway and from Germany by Professor Bartels.

ABSTRACTS

MISCELLANEOUS

Williamson-Noble, F. A. (London).—The reactions of the eye to general disease. *Lancet*, June 18, 25, and July 2, 1932.

"It has, in fact, been my object in preparing these lectures, to sit, so to speak, on the hedge between general medicine and ophthalmology, or—to put it in another way—to convert what might be regarded as a no-man's land between these two departments into an everyman's land." "I have no wish to make my remarks encyclopaedic, and would prefer to be eclectic, picking out from the mass of accumulated data of medical ophthalmology, those features which I think are of interest."

These two sentences from different parts of Williamson-Noble's three Lettsomian Lectures exactly express what he has actually done, and in the doing of it has put the Reactions of the Eye to General Disease into a smaller and sharper focus than usually is the case. Such, at any rate, is likely to be the opinion of most ophthalmologists. If the reader will look up in the dictionary the word "abstract" he will find that it has a number of shades of meaning. For example, it may mean a précis, or, on the other hand, a theft. The reviewer is distracted as to which of these meanings to adopt and must endeavour to combine them, since a précis pure and simple would occupy too much space.

The author first deals with measles which is associated not only with conjunctivitis and corneal ulcers but with non-paralytic convergent squint. "The toxin of measles seems in a fair number of cases to have some peculiar selective action on the fusion centre in the brain, and to bring about concomitant convergent squint, apart entirely from anything in the way of encephalitis." Dealing with the reactions of the iris and ciliary body "which must be considered together," he explains the circulation of the aqueous and the various disturbances which occur in it, makes special reference to the gelatinisation of the fluid, particularly in gonococcal iritis, and to localisation of inflammation to a small area of the ciliary body in syphilitic infection, this leading to gummatous degeneration of the ciliary body and sclera; thus a benign condition (episcleritis) may be simulated when in reality degeneration may be progressing to perforation of the eyeball.

The type of iritis produced by focal infection "usually bears a close resemblance to that caused by the gonococcus. It is subject to relapses, may remain unilateral for a long time, and usually results in the formation of narrow adhesions between the iris and lens, in distinction to the syphilitic type of iritis which is commonly

bilateral and causes broad adhesions." . . . "An interesting feature in many of these cases is paralysis of the sympathetic on the affected side. . . . It is a nice point, and one which has not yet been decided, whether the sympathetic paralysis is the primary cause of the cyclitis. If there are such things as trophic fibres, and if their course to the eye is *viâ* the sympathetic nerves, then interference with these nerves will reduce the resistance of the iris and render it more liable to bacterial invasion." The possibility of endocrine factors taking a share in the causation of chronic iritis and cyclitis is illustrated by a case history. Still dealing with focal infection, what the author has to say about anaphylaxis and elective localisation must be read in the original, but the following may be noted. "No definite opinion seems to have been expressed yet as to whether interstitial keratitis following a blow or injury to the eye can be regarded as attributable to this or not."

A very large part of what the author has to say about the reactions of the iris and ciliary body to syphilis is occupied by his discussion of the Argyll Robertson pupil. Only the opening statement can be given here. "In the first place, what exactly is the Argyll Robertson pupil? It seems to consist essentially in the absence or a marked diminution in the reaction of the pupil to light with preservation of its reaction to accommodation-convergence. There may or may not be associated miosis, and the consensual reflex to light on illuminating the fellow eye may or may not be present. The lesion is to be regarded as the consequence of destruction, whether by toxins, new growth or trauma, of a certain part of the pupillo-reflex arc. It would therefore follow that, although the Arygll Robertson pupil is in a high percentage of cases due to neurosyphilis, it is not necessarily pathognomonic of this disease." Discussing the cerebral connections, the author says, "The special ability of syphilis to produce the lesions is to be attributed to the peculiar affection of the syphilitic toxin for the terminal arborisations of afferent nerves, the toxin spreading from the aqueduct into the subependymal tissues. The localisation of the lesion to this part of the brain may also serve to explain the miosis which is so frequently present, since it is probable that fibres carrying inhibitory impulses from the cortex to the sphincter iridis pass through the periaqueductal tissue, and so are affected by the toxin diffusing into this tissue." (The author indicates that this is only one theory of its localisation.)

The author commences the second lecture by consideration of the lens, reviewing the latest work on its changes in diabetes, and emphasising the necessity, in estimating the utility of the eyes, of remembering to investigate the refraction. Calcium deficiency in connection with the cataract associated with tetany is discussed. Tuberculosis occupies several columns of the *Lancet*. No précis of these could do them justice, but it is worth noting in particular the

author's view on tuberculin treatment. ". . . There is no question in my mind that a number of cases of obstinate infection of the uveal tract and of the cornea do better with tuberculin than with any other form of treatment." Four columns are devoted to high blood pressure. The changes in the retinal vessels are reviewed, with diagrams and references to recent literature. As the author says, "an enormous literature has grown up recently around this subject." Friedenwald's classification of vascular anomalies is given. One or two sentences may be transcribed from what is said on high blood pressure. "The condition of the small vessels passing to the macula may be of great importance in cases of arteriolar sclerosis. In fact, in some cases of vascular disease of the most serious type, these may be the only vessels showing any visible change. (A mydriatic is probably required, and for this purpose cocaine is recommended). The changes observed in these vessels are an alteration in their course, whereby they become corkscrew-like, and an increase in the brightness of their reflex, which renders them like silver wire." Again, "if sclerosis should persist in the retinal arteries over a period of years, the retina itself may begin to show changes . . . Histologically, the change consists in the formation of hyaline nodules in the outer molecular layer of the retina. These nodules are visible with the ophthalmoscope as bright white dots, seldom larger in diameter than one of the main veins. They are usually grouped at the posterior pole of the eye, and may occasionally form a star or a fan-shaped figure round the macula." The discussion of the causes of the appearances in albuminuric retinitis does not lend itself to the making of a précis, or even to the "theft" of isolated parts of it. With regard to diabetic retinitis the author says, *inter alia* "The haemorrhages are for the most part small and circular, owing to their situation in the deeper layers of the retina. There are no cotton wool patches, and the white areas present have a soapy appearance with clean-cut edges. Sometimes they are arranged in a circle round the macula. Finally, there is no retinal oedema to speak of, and there is no star figure." Dealing with the causation of diabetic retinitis here is the opinion given. "It appears, therefore, that arterial sclerosis and renal disease are concomitant, possibly contributory, but not causative of this form of retinitis. . . . The essential lesion in the disease (*i.e.*, the retinitis) has seemed to me to be the recurrence of the deep-seated retinal haemorrhages."

The third lecture hardly lends itself to any form of précis writing. It concerns the retinal changes in anaemia, and the reactions of the optic nerve to disease in other parts of the body and this includes retrobulbar neuritis, papilloedema and optic atrophy. The first named subject (anaemia) must be read word for word in the original. From the others a few sentences may be quoted, notably regarding

retrobulbar neuritis in its relation to disseminated sclerosis. "Its aetiology is still rather a problem. . . . At one time the majority of these cases were attributed to direct extension from sinus disease, but this does not appear to be so. In a series of 76 cases of retrobulbar neuritis, E. D. D. Davis found only 5 in which it was attributable to manifest nasal sinus suppuration, while 14 of the 46 in which a cause could be discovered were due to disseminated sclerosis and 9 to syphilis. Disseminated sclerosis thus seems to be the commonest single cause of retrobulbar neuritis. It is probably commoner than is indicated by Davis's figures, since it may not be until years after the optic nerve involvement that other signs become manifest which would warrant a diagnosis. So that, of the 30 cases in which no cause could be found, it is more than likely that a goodly proportion would turn out to be disseminated sclerosis." Next, one or two references to papilloedema and to optic atrophy. It is pointed out that the pathology of papilloedema is still a subject of discussion and that in some quarters it does not seem to be understood. "The whole thing can be expressed very shortly (diagram given). The subdural space of the optic nerve is normally in connection with the subdural space of the brain through the optic foramen. If, therefore, there is an increase in the general pressure of the cerebro-spinal fluid, such increase will be transmitted to the subdural space of the nerve unless it is distorted in some way at the optic foramen so as to block the communication. The increase of pressure is not, however, transmitted to the interior of the eye. The effect, so far as the optic disc is concerned, is therefore similar to that on, say, the hand when a Bier's bandage is put round the arm, and oedema must obviously occur. . . . Whether the oedema is due principally to blockage of the lymphatics or of the vein does not seem to matter very much." Speaking generally, papilloedema has no localising value. Reference is made to Leslie Paton's findings in this regard.

The author agrees with Leslie Paton that it is futile to attempt to divide optic atrophies into only two classes, and gives some examples.

Lastly, the reviewer may refer to two pitfalls to which the author draws attention in estimating optic atrophy. The first, well known to every ophthalmologist, is the varying physiological tint of the disc. The second refers to the colour as seen by different forms of illumination, and especially the apparent pallor which may be noticed when using an electric ophthalmoscope "particularly if equipped with a new lamp and battery. My own practice in a doubtful case is not to express any opinion until I have had the opportunity of examining the disc through a dilated pupil with a reflecting ophthalmoscope and a yellowish source of light such as is provided by a carbon filament lamp. Even then I like to know

something about the fields and the corrected visual acuity before saying very much."

These scrappy references and quotations require that the reviewer should apologise to the author, though, in the former's favour it may be said that, since nearly thirty columns of closely reasoned matter in the *Lancet* are involved, a book notice would be more suitable and distinctly more easy to write. And will not a book notice ultimately be required?

ERNEST THOMSON.

BOOK NOTICES

Third Report of the Miners' Nystagmus Committee. Privy Council. Medical Research Council. Special Report Series. No. 176. Price, 9d. net.

This report carries a step further the reports of similar but not identically constituted committees published by the Medical Research Council in 1922 and 1923; but that step is a very important one. The earlier committees had arrived at the conclusion that the factor of primary importance in the prevention of Miners' Nystagmus was the supply of adequate illumination, particularly for workers at the coal face. Measures based upon the recognition of this factor have, however, had no success in diminishing the amount which is being paid by coalowners in compensating sufferers from the disorder.

The present report confirms the previous findings as to the importance of adequate lighting if the physical jerking of the eyes is to be avoided, but points out that this feature may never have been noticed, or may have subsided, in many cases in which severe psychological symptoms of the disease exist, while on the other hand nystagmic jerking of the eyes is quite frequently present in miners still engaged in their work and in whom the incapacitating psychoneurotic symptoms may never develop.

The conclusion is reached, and it is a very important advance, that in Miner's Nystagmus we have to deal with a psychoneurosis allied to disabilities of this class in other industries, and to shell-shock. Accordingly, that the affected miner needs to be encouraged to do such work as he is capable of performing, and not allowed, or as at present even practically compelled to remain idle.

That, for patient or observer to concentrate on the cure of the physical manifestation of eye jerking is unjustified, and impedes recovery.

The report ends with the words: "The Committee are strongly of opinion that the practical treatment of the disease from an administrative point of view, should consist in the elimination of a hopeless

dependence on compensation by the provision of opportunities for work of some kind, the end in view being complete restoration to full work underground, under conditions of proper illumination, even if this has to be preceded by a period of work in daylight."

Transactions of the Ophthalmological Society of the United Kingdom. Vol. LII. London: J. and A. Churchill. 1932.

The 52nd volume of Transactions contains the papers read at the congress in Edinburgh last year, with Professor van der Hoeve's Bowman Lecture on "Ocular Movements," together with the report of the Oxford Ophthalmological Congress and the meetings of affiliated societies. It forms a volume of 604 pages and contains much that is of interest. We hope to notice some of the papers at greater length in future numbers. It is to be noted that Professor Priestley Smith and Sir George Berry have been elected honorary members. A new feature this year is the local list of members, which is a very useful innovation.

The council's report shows that the membership of the society is 559, and the balance sheet and treasurer's report shows that the finances are in sound order.

OBITUARY

F. A. C. TYRRELL

FRANCIS ASTLEY COOPER TYRRELL died on New Year's Day in London. He had not been well for some weeks, but his decease following a severe asthmatic attack was quite unexpected. It has come as a shock to his friends, who mourn his loss as that of a hard worker of proved ability and sound knowledge. With quiet unassuming modesty he got through a vast amount of work which was of real value to Londoners, concerned as it was with the ophthalmology of school children.

Tyrrell was 62 years of age. He was a student at Clare College, Cambridge, and at St. Thomas's Hospital, where he held a resident post under Mr. Lawford. He graduated M.B.(Camb.) in 1897, and took the F.R.C.S.(Eng.) in 1901. From 1904-1910 he worked as chief clinical assistant at Moorfields to Mr. E. Treacher Collins, whose memorial service he attended a few weeks before his death. His association with Collins persisted for many years, for, after being oculist to the London School Board, he was appointed one of the visiting surgeons under the Metropolitan Asylums Board in 1905 to the newly started trachoma schools at Swanley and Brentwood, where he attended daily for some years. Brentwood school was given up during the war, but Tyrrell continued as a surgeon to White Oak, Swanley, to the end of his life.

In 1907 he toured America to report upon the treatment of trachoma in school clinics, and visited New York, Toronto, Quebec, and Montreal on behalf of the Moseley Commission. He was the visiting ophthalmic medical officer for the L.C.C. to 29 residential schools and to 6 lunatic asylums. During the war he assisted the ophthalmic department at St. Mary's Hospital, and continued to do so up to recent times. He designed a forceps for the operation of expression and contributed several articles to the Transactions of the Ophthalmological Society. He wrote the account of his grandfather, Frederick Tyrrell, for this journal in the series on British Masters of Ophthalmology (December, 1918), and was related to the famous surgeon, Astley Cooper, whose niece became Mrs. Frederick Tyrrell. His father and brother were also St. Thomas's men, and practised in Great Malvern. His son, just qualified, and his brother-in-law, Dr. Edward Wrench, further strengthen the bond with St. Thomas's. His widow and two children will have received many messages of sincere sympathy from those who knew Francis Tyrrell. Happy in his home and work, of fine appearance and physique, his was an attractive personality, and his memory will live long in the hearts of those who knew him best.

HARRY LEE, F.R.C.S.

THE tragic death of Mr. Harry Lee was noticed in our last number. He was at work, apparently in his usual health on January 11, and had some sort of seizure while driving home from his consulting room.

Mr. Lee was a Yorkshireman, having been born at Dewsbury. His uncle, Dr. Edwin Lee, was a well known physician in Leeds, and it was natural that the nephew should have settled in his native county as he had many friends there.

While at Cambridge, Lee received his blue for Rugby Football, and graduated M.B., B.Ch., in 1910. He pursued his clinical studies at Guy's Hospital, and passed the final F.R.C.S.Eng. in 1911. He was elected surgeon to the Leeds Public Dispensary in 1913, and worked there for 9 years. When Mr. Secker Walker resigned his post on the staff of the General Infirmary, Lee was elected to fill the vacancy, and rapidly acquired a large practice. At the same time he was appointed lecturer in ophthalmology in the University of Leeds. At the time of his death he was senior ophthalmic surgeon to the Leeds General Infirmary.

Lee was an ex-president of the North of England Ophthalmological Society, and succeeded the late Mr. A. L. Whitehead as the Leeds representative on the general editorial committee of this journal.

He was not a prolific author, but his kindness and straightforwardness of manner made him many friends in the ophthalmic fraternity,

while in him, his patients, rich and poor alike, mourn the loss of a distinguished ophthalmic surgeon and genial friend. During the War he served in the British Expeditionary force with the rank of Captain, R.A.M.C.(T.)

CORRESPONDENCE

COLOURED AREAS IN THE SCLEROTIC

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—It happened that immediately after reading Dr. C. H. Usher's excellent article on "Coloured Areas in the Sclerotic," an example of this condition came into my consulting rooms and I considered it might be of interest to report it. It shows many points of resemblance to the cases cited by Dr. Usher, and undoubtedly belongs to this category.

History: Miss G. M., aged nine years, came to me on November 29, 1932, with complaints of some headaches and tiredness of eyes. The visual acuity, left and right, was 6/6 without difficulty. Pupils reacted normally and tension was normal. On drawing aside the lids to examine the conjunctiva, I observed in the right eye a circular area of slaty-blue colour, fairly sharply delimited in all its margins, forming a collar round the limbus, not continuous, but open nasally and temporally. The proximal edge was some 2 or 3 millimetres from the limbus and it varied in depth from 4 to 8 millimetres; conjunctival vessels running over it were enlarged and movable. There were no such appearances in the left eye.

The child was the middle one of three. The irides were of a light green, those of the mother being of the same colour and the father's were reported to be blue. Both fundi were essentially normal, and the right was distinctly richer in pigment than the left. The refraction in both eyes showed a quarter of a dioptre of hypermetropic astigmatism. Hair, eyebrows and eyelashes were dark brown. The mother had never noticed the condition until I drew her attention to it, and therefore one does not know whether the condition is recent or of old standing, stationary or progressive. I shall, of course, examine her from time to time and make observations. There is no nystagmus; there is no history of consanguinity of parents.

This case tends to show, if it shows anything, that the condition can exist without any disability to the visual acuity, or the eyes generally and I fear it casts no further light on the probable aetiology.

Hoping that you will find this of sufficient interest for publication.

Yours faithfully,

D. R. GAWLER.

PERTH,
WESTERN AUSTRALIA.

FISHY VISION

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—Mr. Mayou's further reference to the refraction in fishes raises a number of interesting points. Professor W. A. Osborne, of the Melbourne University, has kindly furnished me with an extract from the work of Hess which reads as follows:—

"The teleosteans are the only animals capable of accommodation whose eyes are focussed at rest for *near* distance. Further they are distinguished from all other animals capable of accommodation in that they have an active accommodation for distance. This takes place through the lens being approximated towards the retina through the contraction of the muscle designated Retractor Lentis by T. A. Beer (formerly called Companula Balleri). I have repeated the researches of Beer in a number of fishes and have been able to confirm his results in the essential points. I found the Periophthalmus, in contradistinction to all other toleosteans previously described, not myopic but emmetropic or slightly hypermetropic."

Handbuch der Normalen und Pathologischen Physiologie. Photoreceptoren. Erster Teil. P. 158, 1929.

I have done no direct work on the eyes of fishes but the work I did on the eyes of mammals and reptiles has made me careful in drawing any conclusions from the action of atropine or eserine. Atropine may paralyse the accommodation in animals; it certainly does so in the primates but we cannot be absolutely sure of its effects in other mammals and I have always checked the result by electrical stimulation of the ciliary muscle when a ciliary muscle is present.

The extract furnished by Professor Osborne makes the problem more complicated than ever. Why should one class of fish have accommodation from the near to the far and another class of fish be emmetropic? I understand that this fish has a periscopic eye which may explain the difference.

The study of accommodation in animals from the cephalopod upwards raises a biological puzzle of a difficult character.

The fish periophthalmus has projecting eyes but, perhaps, hardly periscopic in the strict sense of the word but it spends part of its life out of the water altogether.

Professor Agar, of the Melbourne University, kindly showed me a specimen. It is known, I believe, in many places and certainly on the Queensland coast.

Yours faithfully,

JAMES W. BARRETT.

MELBOURNE.

ACQUIRED MYOPIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—May I add a brief observation to the interesting discussion recently appearing in the ***British Journal of Ophthalmology*** as to the cause of acquired myopia?

It is not, I believe, a routine procedure—it certainly is not in the clinics of the United States—to determine instrumentally the tension and to record the visual fields of young children. This is done usually only in the case of a suspected intra-ocular neoplasm. The reason for the omission of these important diagnostic measures is undoubtedly because they are not considered clinically important. With the instruments ordinarily employed, it is moreover, a difficult and a somewhat fearsome affair for a timid child. The necessity of placing the child on his back in a horizontal position and approaching him with an instrument to be placed upon the eyeballs often causes such a degree of consternation and resistance on his part as to make an accurate determination of the pressure difficult. With the tonometer of Dr. Bailliart, however, after the instillation in the child's eyes of a few drops of butyn, the tension may be determined with the child in a sitting posture, and if done tactfully, is easily accomplished without apprehension on his part and even with his co-operation. If this is done it will be found that an increase of pressure to the extent of from 10 to 15 mm. of mercury is present in a large proportion of eyes in which myopia already exists, as well as in those in which it is developing. Risley pointed out many years ago that the progress of myopia is from the hyperopic eye through the turnstile of astigmatism. Hess has shown that the accommodative effort does not affect the tension in the normal eye; in the astigmatic eye it does.

The points of least resistance in the young elastic structures are at the anterior and posterior poles. Before and at about the advent of adolescence all of the tissues are undergoing important changes. The endocrines are beginning to take on increased activity. If the balance is not maintained in the functioning of the thyroid, the pituitary and the sex glands, an increase of tension will often manifest itself in the eyes. That the increased pressure does not become more evident is due to the fact that compensation is found in the stretching of the eyeballs. As the elasticity gives way the pressure is reduced. It is again increased as the progress of the myopia ceases. In malignant myopia these changes are continuous. It will usually be found that each increase of approximately one dioptre of myopia will be preceded by a hypertension varying from 10 to 15 mm. Hg. This, of course, will be variable depending upon the resistance of the tissues.

Again, when the middle period of life is passing and when functional glandular changes are diminishing or ceasing, especially during a time of mental or physical stress, the intra-ocular balance is again disturbed. The sclera and the cornea by the 50th or 60th year will have become denser. Then the point of least resistance is not at the poles of the globe; it is in the cribriform plate of the optic disc and when hypertension occurs a cupping of the disc results. Occasionally it happens that in an adult, for one reason or another, such as continued depressed vitality or low blood pressure, the body tissues remain less resistant or are not sclerosed. They then may respond to an increased intra-ocular pressure precisely as they do in childhood.

The following typical cases will illustrate each of these forms.

A girl of ten years comes because of an increased myopia.

O.D. minus 3·00 D. sph. V. equals 5/8.

O.S. minus 1·50 D. sph. V. equals 5/8.

Tension in the right eye, 28; in the left, 26 (Bailliart).

Slight congestion of both optic discs and narrowing in all directions of visual fields, headache but not severe in character. The diminished visual acuity and restriction of fields is due in all probability to the pressure on the optic nerve fibres as they leave the disc. The child is treated with pilocarpine each night on retiring and the emotional life studied, both of which are important.

If the child can be safely carried over the period in which these deep bodily changes are taking place the structures will themselves become so adjusted that further progress of the disease does not take place, and under correction normal acuity returns.

Toward the end of middle life similar disturbances may occur. A case will illustrate the development of myopia produced by hypertension in an adult rather than the classical symptoms of glaucoma.

A man of 46 occupying an important official position with much responsibility, a solicitor, and having low blood pressure, suddenly within a period of a few days complained of having lost the sight in the left eye. Prior to this attack he had had for a number of years a myopia of about three dioptres in each eye and a moderate lateral astigmatism and with practically normal acuity. His refraction was found to be as follows:

O.D. minus 2·75 D. sph., minus 0·50 D. cyl., axis 105°, V. equals 9/8.

O.S. minus 6·25 D. sph., plus 1·37 D. cyl., axis 135°, V. equals 7/8.

Tension in the left eye, *21 mm. Hg*; *left eye, 42 mm. Hg*.

There was present a definite narrowing of the left field, but no cupping of the discs.

The higher tension in the eye with the higher myopia would indicate that an increase of intra-ocular pressure had resulted in a posterior polar relaxation, the scleral tissues being less resistant than is commonly found in late middle life. The hypertension therefore, produced an increased myopia instead of cupping of the disc.

Intra-ocular hypertension is a symptom perhaps rather than a primary cause of myopia and it is probably not the only reason for the posterior relaxation of the globe. In a family of four children, the oldest and the youngest male and female who are tall, fair and similar temperamentally and physically, have developed a fairly high degree of myopia. The second and third who are shorter and sturdier, living under like conditions, have normal eyes. They form two groups having dissimilar likes and aptitudes. The former have in all probability potentially structural tissue weaknesses which the second group escapes.

Do not such cases as these give an impression that progressive myopia may be a temporary form of glaucoma simplex juvenilis? The recognition of this aetiological factor should have an important bearing on our method of managing this serious disease in children.

Yours faithfully,

PARK LEWIS.

BUFFALO,
NEW YORK.

NOTES

Death

THE death is announced of Professor Landolt of Lugano.

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Appointment

MR. CECIL W. R. MCCALDIN has been elected Hon. Assistant Surgeon to the Nottingham and Midland Eye Infirmary.

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Royal London Ophthalmic Hospital, Annual Dinner

THE Annual Dinner of past and present students of the London Royal Ophthalmic (Moorfields) Hospital, was held at the Langham Hotel on Thursday, February 9.

Sir Arnold Lawson was in the chair, and among the sixty-seven present were:—Lt.-Gen. Sir H. B. Fawcus (Director-General of the Army Medical Service); Air Vice-Marshall J. McIntyre; Sir William and Lady Lister; Lady Lawson; Mr. T. W. Luling (Chairman of the Hospital); Dr. A. M. H. Gray (Dean of the Medical Faculty of the University of London); Sir W. Morley

Fletcher; Professor Woollard; Dr. Brinckworth; Dr. Russell Brain; Messrs. A. F. MacCallan, M. L. Hepburn, Foster Moore, R. Affleck Greeves, Frank Juler, Charles Goulden (Dean of the Medical School), M. H. Whiting, Humphrey Neame, W. Duke-Elder; Dr. Ida Mann.

The Dean, in response to the toast of the Hospital and Medical School, stated that the Medical School was in a flourishing state. During the year 1931-32, one hundred and seventeen students had attended the practice of the Hospital and of these sixty-seven were new students. Three hundred and twenty-eight lectures had been given. The School still prepared by far the largest proportion of students who sat for the Diploma of Ophthalmological Medicine and Surgery. During the past year, of thirty-one candidates who were successful in the first part of the examination for this Diploma, twenty-one received their instruction at Moorfields, and of the thirty-one who were successful in the second part, twenty-five were students of the Hospital.

Rebuilding of the Out-patients' Department and the Medical School will begin in March, and when completed, facilities for clinical instruction and laboratory teaching will be greatly improved.

FUTURE ARRANGEMENTS

1933

March 3.—North of England Ophthalmological Society, at Liverpool.

March 10.—Royal Society of Medicine, Section of Ophthalmology (Clinical Meeting).

March 31.—North of England Ophthalmological Society, at Sheffield.

April 4.—Midland Ophthalmological Society, at Oxford Eye Hospital.

April 17-21.—XIV International Ophthalmological Congress, at Madrid.

May 11-13.—Ophthalmological Society of the United Kingdom, Annual Congress, at the Royal Society of Medicine.

May 26.—Midland Ophthalmological Society, at Gloucester Royal Infirmary.

June 9.—Royal Society of Medicine, Section of Ophthalmology (Annual Meeting).

July 5-8.—Oxford Ophthalmological Congress, at Oxford.

October 6.—Midland Ophthalmological Society, Annual Meeting, at Birmingham and Midland Eye Hospital.

December 5.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *January, 1933.*

PILLAT. Changes in the eyeground in Wilson's disease (pseudosclerosis).
DE SCHWEINITZ and COWAN. An unusual corneal lesion, probably congenital and familial in character.
LEPARD. Lipemia retinalis.
MOTTO and DUNNAVAN. Hemangioma of the conjunctiva.
BERENS and POSNER. The circulation of the intraocular fluid.
PARKER. Bilateral facial spasm.
FOLK and TAUBE. Vaccinia of the eyelids and conjunctiva.
PFINGST and TOWNES. Endarteritis obliterans with spontaneous gangrene of both corneae.

Archives of Ophthalmology. *December, 1932.*

ELLIOT. Trephining.
MARSH. Lenticonus posterior: further study.
STOUGH. Choking of the optic discs in diseases other than tumour of the brain.
MACMILLAN. Transplantation of the lacrimal sac in chronic suppurative dacryocystitis.
REDWAY. Antiquity of the forms of the transparent ocular media.
KLIEN-MONCRIEFF. An atypical pigment line of the cornea: a biomicroscopic and histological study.
GOLDSTEIN and WEXLER. Retinal vascular disease in a case of acute lupus erythematosus disseminatus.
MOTT. Muscle recession with tendon stump and Tenon's capsule fixation.
MEEK. Sarcoma of the iris: report of a case complicated by a cyst.
KRUG and SAMUELS. Venous angioma of the retina, optic nerve, chiasm and brain: a case report with postmortem observations.
O'BRIEN. Experimental cataract in Vitamin G deficiency.

January, 1933.

NAFFZIGER. Pathologic changes in the orbit in progressive exophthalmos, with special reference to alterations in the extra-ocular muscles and the optic discs.
FINNOFF. A syndrome in uveal tuberculosis.
PILLAT. Production of pigment in the conjunctiva in night blindness, prexerosis, xerosis and keratomalacia of adults.
O'CONNOR. Early grafting in burns of the eye.
CASTEN and SHAAD. Diagnostic value of tests of the light sense in glaucoma.
KAUFMAN. Morgagnian cataracts and their complications, with a report of a case of spontaneous rupture of the lens capsule causing secondary glaucoma.
MCKEOWN. Detachment of the retina: Guist operation and report of cases.
KATZ and DELANEY. Dystrophia adiposa corneae.

Annales d'Oculistique. *December, 1932.*

CHAILLOUS. J. Babinski. 1857-1932.
ROY. Foucher. 1856-1932.
CHIAZZARO. Ocular infections with anaerobic microbes.
TERSON. Three little known portraits of masters of olden days.

Archives d'Ophtalmologie. *January, 1933.*

Report of the Annual Meeting of the Society for the Prevention of Blindness.
BURGHGRAEVE. Anatomico- pathological study of certain ocular lesions following infection by *Treponema cuniculi*.

Revue Générale d'Ophtalmologie. *June, 1931-1932.*

GOURFEIN-WELT. Sympathetic ophthalmitis.

Klinische Monatsblätter für Augenheilkunde. *January, 1933.*

SCHIECK. The use of autogenous blood injections into the anterior chamber in tuberculosis of the anterior half of the eye.
ENGELKING. Colour weakness and artificial colour blindness.
BUNGE. The duration of the result of cyclodialysis.
KURZ. On keratoconus and its treatment,
BIERINGER. Myopic extraction in an eye with successful keratoplasty.
ZYKULENKO and WELTER. Transparent healing of a corneal transplantation in a traumatic leucoma.
PAUL. Drawing of fundus pictures.
HERZAU. On osteomyelitis of the frontal sinus in three children of one family.
HOUWER. On the question of the clinical diagnosis of angioma of the choroid.
OLÁH. Rosacea iritis.

MÉSZÁROS, and ZOLTÁN-TÓTH. On the peripheral vascular system in glaucomatous subjects.
ILLES. On X-ray therapy of episcleritis.
PAVIA. Cinematography of the living fundus oculi.

Zeitschrift für Augenheilkunde. *January, 1933.*

HAMBURG. On the beneficial influence in tabetic optic atrophy of raising of cell oxydation.
COMBERG. A new electrical ophthalmoscope for use with the direct and indirect method.
STERN. On entoptoscopy and its clinical uses.
JUNGRAW. A fatal case of endophthalmitis and purulent meningitis from an injury to the cornea with a knife.
LAUTERSTEIN. *Ophthalmopathia trigeminociliaris.*

Annali di Ottalmologia e Clinica Oculistica. *July, 1932.*

HARRUGA. Experimental adhesive choroiditis.
VANCEA. Primary post-traumatic tuberculosis.
GIUFFRIDA. On the prevention of industrial eye injuries.

Bollettino d'Oculistica. *December, 1932.*

ALAJMO. On the aetiopathogenesis of familial degeneration of the cornea.
STENICO. Initial experiments directed to the discovery of histamine-resembling substances in the aqueous of glaucomatous patients.
FEDERICI. On a particular recently described form of chronic pseudo-membraneous conjunctivitis.
BELLORA. A case of nodular tuberculosis of the subcutaneous tissue of the lids.
FOCOSI. On a case of mycotic ulcer of the cornea from Cephalosporium.
ANELLI. Pseudo-vaccinial pustule of the lower right lid produced by tetragenes.

January, 1933.

BIETTI. On a little known form of annular pigmentation of the posterior pole of the lens associated with the deposit of pigment on the posterior surface of the cornea.
PANICO. The influence of pterygium on corneal refraction.
DE VINCENTIIS. Three cases of leucosarcoma of the uveal tract.
DENTI. The cure of detached retina by Gonin's operation.

Revista Oto-Neuro-Oftalmologica. *May-June, 1932.*

CAVINA. The present state of pituitary surgery.

Revista de Ophtalmologia. *December, 1932.*

BURNIER. *Ectopia lentis.*
GOMES. Lesions and war injuries observed during the civil war.
ALVARO. Illumination
BUSSACA. Ocular incidents in the treatment of malaria.
MATTOS. Retinography.
DE REGENDE. Frequency of ocular phlyctens with regard to age and sex.
ALVARO. Pterygium in St. Paulo. Statistics.

Archivos de Oftalmología Hispano-Americanos. *January, 1933.*

MÁRQUEZ. The blepharoplastic operations of the Basque surgeon Argumosa.
MATA. The practice of autotherapy in scrofulous ophthalmia.
ENRÍQUEZ. Mechanical excitation of the retina.
RODA. Poulard's stricturotomy.
AMAT and ENCISO. Cocaine in ocular therapy.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica. *February, 1933.*

PAVIA, DI CIO and DUSSELDORP. Cinematography of the fundus of the eye. Diagnosis of cranial hypertension by means of cinematography of the fundus.

Revista Cubana de Oto-Neuro-Oftalmiatria. *Nov.-Dec., 1933.*

VELHAGEN. On the formation of a retinal tear before detachment.
ROLLET and MULLER. On the influence of climate and geography in the spread of trachoma.
GONZALEZ. Loss of visual orientation following cataract operation.
POYALES. Pre-operative, operative and post-operative complications of cataract extraction.
CESPEDES and GARCIA. Electrolysis in narrowing of the lacrimal passages.
ESTEBAN. Fibrillary contractions of the orbicularis.
——— Iridocyclitis following tattooing.